THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING
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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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PRELIMINARY REPORTS OF SOME OF THE DISEASE AND PEST PROBLEMS OF KENAF, HIBISCUS CANNABINUS L., IN SOUTH FLORIDA[1]

INTRODUCTION

Current world conditions have raised some uncertainties as to the continued adequate supply of the so-called soft fibers such as jute. The fiber produced by the kenaf plant, Hibiscus cannabinus L., has long been recognized as a good substitute for jute for many uses. It has not been grown commercially in the temperate zone, although considerable production has occurred in the subtropics.

Until recently culture of kenaf in the United States on a commercial basis has not been attempted because of competition with more cheaply produced fibers from areas of relatively abundant hand labor. Now, however, attempts are being made to produce the fiber in south Florida, using mechanical methods of production.

Large acreages of the plant were grown during the past summer in an area bounded by Vero Beach in the north to Belle Glade in the west and Fort Lauderdale to the south. A total of 2500 acres was planted. The conditions described herein were noted throughout this area. Approximately 750 acres of the 2500-acre total were used for fiber production. This was grown on the muck soil of the Belle Glade area. The remaining 1750 acres were grown for seed increase on sandy soils of the east coast.

When a crop is introduced into a new area of culture a varying length of time usually elapses before a large number of problems are noted on that crop. This has not been the case with kenaf in south Florida since in its first year of extensive planting it has suffered severely from many diseases and pests. It is the purpose of this report to enumerate and describe some of the troubles encountered this past season.

I - SOME PATHOLOGICAL PROBLEMS

By Warren N. Stoner[2]

The first commercial planting of kenaf for fiber production was made on July 8, 1951, at Hillsboro Plantation, Belle Glade. Good germination ensued and rapid growth occurred. At the end of a month abnormalities were observed in the plants, which were 12 inches in height. The youngest developing leaves were mottled severely and presented the typical mosaic pattern associated with many of the non-persistent viruses. Green, raised, blister-like areas were interspersed in lighter green tissue over the entire lamina of the leaf. This mottling was not confined to the interveinal area (Fig. 1B). The plants thus affected were definitely stunted. Internodes at the tip were shortened, giving the plant a rosette appearance. Many of the affected plants died. The condition was general throughout one edge of the planting at the time of first observation. Subsequent observation proved that the condition was spreading, and the spread-pattern and distribution seemed to indicate a winged insect vector. Field specimens were taken at this time for laboratory examination.

Portions of affected plants were surface sterilized and placed in damp chambers. After three weeks the cuttings had formed new roots and were free of any organism. Repeated microscopic examination of all portions of the affected plants failed to disclose any fungi or bacteria consistently associated with the condition.

By this time leaf malformation in the field plants had become more severe (Fig. 1C). The mottling first observed was not so strong and the plants were making a fair growth. A twisting of the distal third of the main stems was noted. Fresh material was taken from the fields and sap inoculations were made by the carborundum method of Rawlins and Tompkins (6) to ten plants each of the following species: kenaf, Hibiscus cannabinus L.; Zucchini squash, Cucurbita pepo L.; Turkish tobacco, Nicotiana tabacum L.; Nicotiana glutinosa L.; okra, Hibiscus esculentus L; and purple thornapple, Datura tatula L. Six days after inoculation a definite mottling was seen in four of the ten kenaf plants, but no symptoms were visible in the other species. One-hundred percent checks were maintained on this series and all remained normal. All plants in this series were held in the greenhouse to maturity and the only abnormality observed was in the four kenaf plants originally showing the mottling. They were stunted in growth, compared to the checks, and failed to set seed capsules. A second mechanically inoculated series developed the same symptoms in 12 of 20 inoculated kenaf plants. Globe amaranth, Gomphrena globosa L., and Zinnia elegans Jacq. were included in this series but did not develop any symptoms. The transmission of the condition

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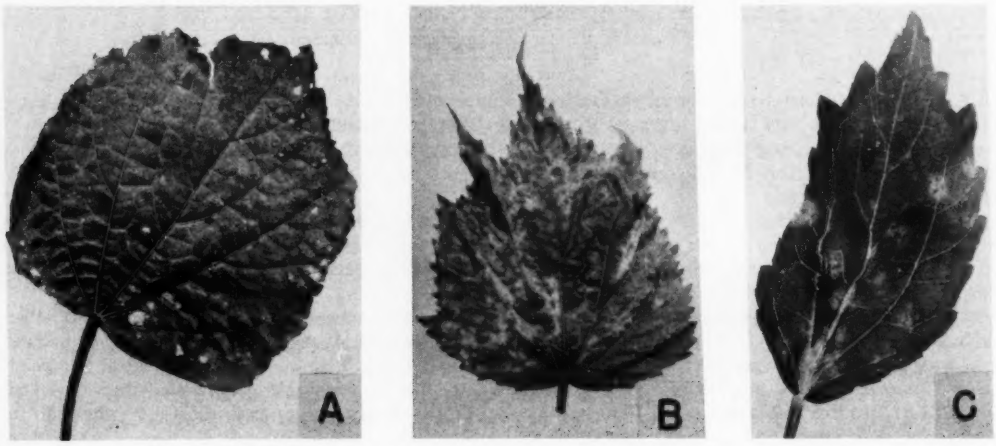

FIGURE 1. Kenaf leaves. -- A is normal, note heart shape and comparatively smooth margin. B is affected with virus-like condition, note mottling, green blistered areas, and irregularity of margin. C, later, more severe malformation, but note mottling is absent. Whitish spots on C are infections of Leveillula taurica (Lév.) Arn. All photos by Grant E. Averill. (x 3/4).

FIGURE 2. Three foot sections of the tips of mature kenaf. -- A normal, note seed capsules located at axils of leaves and smoothness of stem. B (center two specimens) plants suffering from virus-like condition. Note absence of leaves, roughness of stems, witches-broom caused by vegetative growth of axillary buds, and seed capsules on axillary shoots. C is stalk suffering from the virus-like condition which has shed all leaves. The axillary shoots have died and shed. Photo by Grant E. Averill.

from kenaf to kenaf indicate that a virus infection may be present.

Several varieties of kenaf in the breeding nursery at the Everglades Experiment Station have been examined and approximately 80 percent of these varieties have the suspected virus infection on the basis of mottling, leaf malformation and stem twisting symptoms. Species related to kenaf in the genera Hibiscus, Urena, Kosteletzkya, growing in the same nursery are apparently normal.

As the commercial planting began to mature other symptoms began to develop and a fungus, Colletotrichum hibisci Polacci[3], could be consistently associated with these symptoms after high winds and heavy rains on October 2, 1951. This greatly complicated investigations, and whether or not the following field symptoms are attributable to a virus or the fungus is now under further study. Maturing plants became increasingly stunted owing to a shortening of the internodes at the tips. The axillary buds were forced into growth, and young leaves of the main shoot and the axillary sprouts developed an internal darkening of the vascular strands in the laminae and leaf petioles, extending into the main stem. This was followed by a necrosis and die-back of the leaf tissue and further stimulation of side shoot development (Fig. 2B). Some fasciation of the main stems was observed at this time and the plants had a witches-broom appearance. In many instances the axillary shoots died and leaves on the entire stalk dropped, leaving a naked straight stem dying back at the tip (Fig. 2C). This leaf drop may have been influenced by a very severe infection of Leveillula taurica (Lév.) Arn. (2). (Healthy plants grown from checks developed this disease in the greenhouse and shed the heavily infected leaves.) When seed capsules began to develop the previous stimulation of the axillary buds of the main stem prevented flowering, so that the stems were entirely bare or formed flowers only on the weak axillary shoots instead of in the axils of the main stem as is normal (Fig. 2, B and C). Occasional stem lesions of the fungus C. hibisci were found later in the season but were considered only incidental.

It has not been possible, to date, to associate any particular insect with the conditions described, but preliminary tests are being made with various insect species found in kenaf fields to try to determine if there is a relationship. The cotton or melon aphid, Aphis gossypii Glover, is suspected as the most likely vector, in case an insect vector is involved, since this species was present in large numbers and became distributed in the fields in much the same pattern as the disease.

Investigations of these pathological problems of this potentially valuable crop are being continued.

II - OTHER PATHOLOGICAL PROBLEMS

By Frank V. Stevenson[4]

Abnormalities described in the first section of this paper were accompanied by other diseases, which occurred in varying degrees of severity. Disease symptoms were as numerous and varied as the different organisms involved would indicate. Many plants were heavily attacked by several organisms, some of which tended to overgrow the others. The abundant growth of several common organisms which are widespread and which have a wide host range in plants already grown in the area under consideration is worthy of emphasis.

Prominent among these were one or more species of Fusarium which caused wilting and subsequent death of plants at several widely separated locations. This disease was very severe in a field that was last planted to tomatoes grown on beds. The location of the beds was marked across the field by alternating swaths of killed and healthy kenaf plants spaced regularly by the width of the former tomato beds. Fusarium was obtained in cultures from the roots to the tip of the plant and presence of spore masses was quite apparent over the whole plant.

A stem blight of severe degree has been tentatively ascribed to the attack of Sclerotium bataticola Taub. It caused silvery dark brown watersoaked lesions which eventually blackened. It caused death of many plants in large circular areas in the field when they were quite immature. A possible association with insect transmission is discussed in section III of this paper.

An Ascochyta leaf blight and a species of Rhizoctonia also were present. Bacteria were found but did not seem to be of primary importance. There was, in addition, an anthracnose condition showing salmon pink spore masses on collapsed tips of the stem and in the axils of the capsules. These conditions are treated more fully in the first section of this paper, as is one due to a powdery mildew.

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[3]The author is indebted to John A. Stevenson of the U. S. D. A. for determination of this fungus.

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FIGURE 3. Hippopsis lemniscata (Fab. resting on diseased stems, showing possibilities of contamination with an organism tentatively identified as Sclerotium bataticola Taub., prior to nocturnal feeding and ovipositing. Photo by Grant Averill.

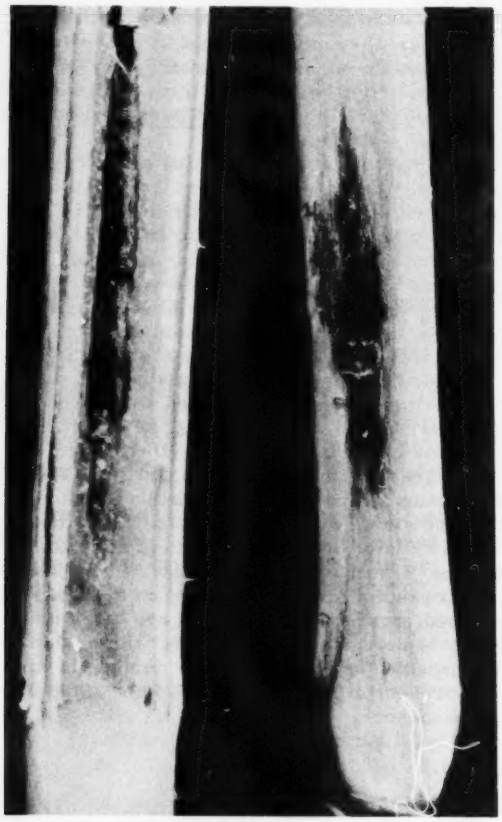

FIGURE 4. Enlargement of section of kenaf stem showing lesions extending from point of oviposition of Hippopsis lemniscata (Fab.); stem split to show deep penetration by the organism tentatively identified as Sclerotium bataticola Taub. The egg puncture shows as a ringed area on cut out section of stem at right. Photo by Grant Averill.

III - HIPPOPSIS LEMNISCATA (FAB.) (COLEOPTERA: CERAMBYCIDAE) IN RELATION TO KENAF DISEASES

By William G. Genung[5]

A general consideration of the pathological conditions in kenaf plantings in southern Florida must include the possible role of insects as mechanical vectors in the transmission of fungus diseases. Field observations strongly indicate that such an implication exists.

Genung (3) has mentioned the possibility of transmission of a fungus disease tentatively identified as charcoal rot, Sclerotium bataticola Taub., by the Gerambycid, Hippopsis lemniscata (Fab.), in this crop. The habits of this beetle would seem well adapted to aid in the dissemination of the causal organism of the disease. The discolored, diseased stems are the favored diurnal resting place of adults, which feed upon and oviposit in healthy tissue at night (Fig. 3). Many stem lesions seem to originate from these points of activity (Fig. 4). The beetles could carry the organism on their bodies and implant the disease in healthy tissue, or the wounds produced may become infected in other ways. In young plants the condition associated with attack by this beetle is usually fatal.

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is usually fatal.

A pith rot also seems to develop in many cases and may be initiated by these beetles through oviposition activity. In oviposition the ovipositor is pushed through a hole gnawed in the wood and the egg is deposited in the pith. In the case of pith infection there is a liquefaction that is often sufficient to drown the young larvae. The plants usually survive this condition but the pith is gradually destroyed and the stem becomes a hollow tube for most of its length. Counts made in three fields showed in each instance about two and one half times more infection in stalks that had been infested with cerambycids than in stems where no beetle infestation could be noted.

In consideration of the abundance of H. lemniscata in kenaf plantings in south Florida during 1951, it is of interest to note that the species apparently has not previously been recorded as a pest of any important crop plant. The principal host in the Everglades area has been ragweed. Lighter infestations of this cerambycid occur in Bidens sp. and in Amaranthus sp. It now appears that well timed weed control will be an important consideration in prevention of heavy infestation in kenaf. An excellent kill of adults was obtained with an aerial application of 5% DDT dust at 30 pounds per acre in a commercial planting.

A record has been kept of insect species occurring in kenaf plantings. It was felt that inclusion of the list in this report might be of interest. Some of these notes antedate or precede observations made on Hippopsis lemniscata as a possible vector of charcoal rot and a pith condition in kenaf. Species encountered were: Southern green stink bug, Nezara viridula L.; cotton aphid, Aphis gossypii Glov.; cotton stainer, Dysdercus suturellus (H-S); big thighed bud, Acanthocephala spp.; leaf footed bug, Leptoglossus phyllopus (L.); large milkweed bug, Oncopeltus fasciatus (Dall.); potato leaf hopper, Empoasca fabae Harr.; American grasshopper, Schistocerca americana (Drury); Schistocerca aleutacea rubiginosa Scudder; red legged grasshopper, Melanoplus femur-rubrum propinquus Scudder; Chortophaga viridifasciata australior R. & H.; field cricket, Gryllus assimilis Fab.; granulate cutworm, Feltia subterranea Fab.; Southern armyworm, Prodenia erdania (Cram.); saltmarsh caterpillar, Estigmene acraea (Drury); Hippopsis lemniscata (Fab.); Leiopus spp.; Diabrotica undecimpunctata howardi Barber; Hippodamia convergens Guer.; Scymnus spp.; Zelus bilobus Say.

Several species that have not been determined are not included in this list.

IV - NEMATODES

By Walter H. Thames, Jr.[6]

Watson and Bratley (8) reported that Hibiscus cannabinus L. seemed very susceptible to root-knot which caused much stunting of the stalks. Examinations of kenaf plants brought in from 1951 plantings at the Everglades Experiment Station, from experimental plantings near Lake Worth, and from commercial plantings in south Florida have shown moderate to very severe infestations of the root-knot nematode, Meloidogyne incognita (Kofoid & White) Chitwood. Infested plants in the fields are generally stunted or retarded in growth. Stems and terminal leaves show symptoms similar to those produced by nitrogen deficiency, particularly on sandy soils.

Observations of plants from the Everglades Station indicate the presence of a nematode-fungus complex. Preliminary studies have not yet shown damage typical of the meadow or awl nematodes.

V - AGRONOMIC SIGNIFICANCE OF DISEASE AND PEST PROBLEMS

By Charles C. Seale and Edward O. Gangstad[7]

The virus-like condition mentioned in section I, although not noted in previous years, has been the most serious disease problem of kenaf this past season. The disease was observed throughout the general area of production in Florida. The rapid and widespread development of this condition indicates that the source of infection either is present in natural vegetation of the area and has built up in the kenaf plantings or has been introduced in seed. This diseased condition appears to have developed independent of location or age of plant. The most severe symptoms have been found under conditions of high natural soil fertility or where application of commercial fertilizer was relatively high. It has become more widespread as the season progressed. There has been a tendency for plants not severely affected to show signs of recovery. When this disease was severe, yields of seed and fiber were substantially reduced.

Injury by Fusarium wilt has been relatively severe at several locations and may result in a complete loss of the crop where the soil is heavily infested with inoculum from refuse of a previous susceptible crop. Injury from other fungus diseases has been limited in area and degree but represents a hazard to crop production.

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Infestation by the Cerambycid beetle, noted in section III, reduces the yield of fiber and seed through retardation of growth, lodging and internal deterioration of the stalk. Injury from other insect pests has been relatively light, but under some conditions may become serious. Some of these insects may be involved in transmission of diseases.

Nematode injury has reached serious proportions on lighter sandy soils, resulting in a complete failure of the crop at several locations. Nematode infestations are often associated with Fusarium wilt, particularly in areas where vegetable crops are grown.

Fusarium wilt and nematodes have been noted to be the most serious problems in commercial production of kenaf by Horst (4) in Java and Seale et. al. (7) in Florida.

Observation on the first year's commercial plantings have confirmed the seriousness of nematodes and Fusarium wilt. However, this year the suspected virus disease has proved to be the most widespread and serious pathological problem with the commercial variety now planted.

VI - POTENTIAL SOURCES OF DISEASE RESISTANCE

By James B. Pate[8]

This past summer severe root-knot nematode and Fusarium wilt infections developed in the kenaf breeding nursery on peat soils at Belle Glade and in a disease nursery on light sandy soils near Lake Worth. Mosaic symptoms indicating a possible virus disease also were present in the breeding nursery.

The strain of kenaf used in commercial plantings in Florida was originally obtained from El Salvador and has been grown commercially in Cuba. According to Crane and Acuna (1), it is a mixture of the viridis and vulgaris varieties, two of the seven varieties of kenaf identified by Howard and Howard (5). Nematode and wilt ratings obtained at both nursery locations show that the commercial variety and closely related types are very susceptible to both nematodes and wilt. Several Hibiscus ornamentals are very resistant. They appear to be the most promising sources of resistance to nematodes and wilt. A strain of Hibiscus sabdariffa L. from French Equatorial Africa is also very resistant to nematodes, though susceptible to wilt.

In the virus-like condition the source of resistance is not as well defined at present. In the case of nematodes and wilt the commercial variety is quite susceptible. Kosteletzkya althaeifolia (Chapm.) A. Gray, Urena lobata L., Hibiscus grandiflorus Michx., several strains of Hibiscus sabdariffa L., and certain Hibiscus ornamentals have not shown the symptoms. Some strains related to the commercial variety also may be potential sources of resistance. These materials must be further evaluated before resistant types can be selected.

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[8]Research Agronomist, Division of Cotton and Other Fiber Crops and Diseases, U. S. Bureau of Plant Industry, Soils and Agricultural Engineering.

WATER MOVEMENT IN TOBACCO PLANTS AFFECTED BY BLACK SHANK

Harry R. Powers, Jr.

In tobacco plants affected by black shank (Phytophthora parasitica var. nicotianae), local lesions develop in the roots and basal portions of the stems. The severe wilt symptoms that follow are suggestive of those which generally occur in plants with systemic, vascular infections. This sudden wilting of black shank-infected plants has been attributed to the effect of a toxic metabolic product of the pathogen.[1]

Recent experiments indicate that wilting is caused by the inability of the xylem in the affected portion of the stem to function in water transport. This has been demonstrated by three experimental procedures using plants inoculated at the base of the stem. A severely wilted, diseased plant was removed from soil and the lower portion was imersed in water. While under water, the stem was cut off just above the lesion and the freshly cut stem of the top of the plant was kept in water. After two hours the wilted top had regained its turgor. Plants treated similarly, with the exception that the stem was cut off below the lesion, failed to recover regardless of how long they remained in water. A second type of experiment was conducted to determine whether a diseased plant would recover while the pathogen was active within its tissues. A waterproof paper cup was fitted around the stem of a diseased plant above the lesion. The cup was sealed at the bottom with melted paraffin. The cup was filled with water and a notch cut into the stem below the water level. In this case the plant also regained its turgor. The third procedure was to place the roots of both diseased and healthy plants in an aqueous solution of eosine dye. Within four hours the dye was found to be distributed throughout the healthy plant while in the diseased plant it had not moved past the lesion.

These observations indicate that wilting is due to local obstruction of water movement through the vascular elements rather than to systemic effects of toxic metabolic products of the pathogen. This, however, does not preclude the possibility that toxic substances are produced and are in some way responsible for this obstruction. Histological studies are now being made to determine the internal condition of the vascular elements which may afford further information as to the actual cause of wilting in plants affected with black shank.

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VIRUS DISEASES AND INTERESTING VIRUS-LIKE SYMPTOMS IN MELONS IN ARIZONA - 1950-1951

Paul D. Keener

In a previous report (1), the occurrence of a virus-induced mosaic in cantaloupes and honey dew melons resulting in symptoms ranging from scarcely perceptible to deep yellow mottles (Fig. 1), was noted. One of the viruses involved was found to be readily transmitted by the juice-carborundum-rubbing technique, as well as by the pea aphid, Macrosiphum pisi Kalt., to squash, cantaloupe and honey dew seedlings. In honey dew melons in addition to yellow mottles, the virus involved causes alterations in the shapes of leaves as well as the leaf surfaces (Fig. 2, A). The shapes and surfaces of fruits are frequently abnormal (Fig. 2, B). A virus occurring in the seeds of squash (in as many as 14 percent), available to growers on the market, was also found to be capable of infecting seedlings of cantaloupes and honey dews, resulting in similar symptoms (i. e., in faint to brilliant yellow mottles, and distortions in leaf and fruit shapes and surfaces). During the period 1947-49, the percentages of infected honey dew plants in the field were high, but they did not compare with the recent severe infections of 1950-51. In the Deer Valley previous to 1951 effects of mosaics were mild in both cantaloupes and honey dews and the percentages of infected plants were low. In 1951, many fields showed as high as 100 percent of the plants affected. In addition, much fruit damage was noticeable. Unfortunately, many of the heavily infested honey dew plantings were in close proximity to plantings of alfalfa. Alfalfa is a good increase crop for the pea aphid, Macrosiphum pisi.

Field surveys for the past five years indicate that the 1950-51 mosaic infections in both cantaloupes and honey dews in the Deer and Salt River Valleys were the heaviest and most widespread in several years. The increased acreages devoted to honey dew plantings pose a serious problem for melon growers. Seed germination tests at this station show that higher percentages of seeds of certain commercial lots of squash and honey dew melons are "apt to carry" mosaic viruses than is the case with cantaloupes. Also, the trend in 1950-51 was for more seeds to be infected than in previous years. In greenhouse tests at Tucson, percentages of infected cantaloupe and honey dew melon seedlings, from commercial seed lots grown under caged conditions, are indicated in Table 1.

Table 1. Incidence of seed-borne mosaics in cantaloupes and honey dew melons, 1949-1951. Greenhouse trials.

Year	Percentages of infected seedlings	
	Cantaloupe	Honey dew
1949	0.5	2.5
1950	None	1.4
1951	0.6	3.4[a]

[a]These seedlings were grown from identical lots of commercial seeds used by growers in the Deer Valley plantings during 1951 when field infections varied from 80-100 percent, at harvest time.

The same trends between seed infections and percentages of diseased seedlings were noted in the field in experimental plots at Tucson. The seedlings were examined just after emergence. These plots were isolated from other melon plantings but were adjacent to areas of alfalfa. The percentages of field infections are shown in Table 2.

The proximity of plantings of honey dew melons to such aphid-inhabiting crops as beets, carrots, alfalfa, etc., seems to have considerable influence on the amount of mosaic present as well as the rapidity of spread and distribution within the planting. In 1950, a commercial planting of honey dew melons adjacent to carrots and alfalfa on one side with highways on the others, exhibited most of the mosaic on the side in proximity to the carrots and alfalfa. On the carrot-

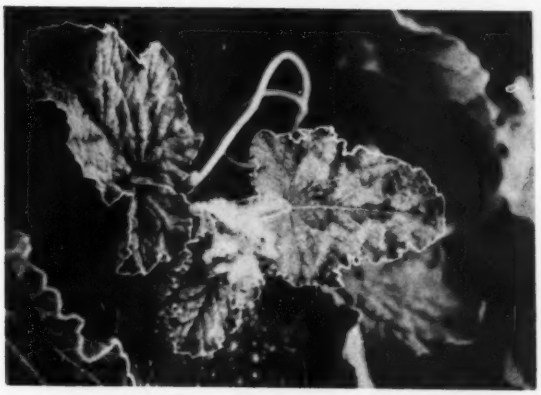

FIGURE 1. Yellow mottle symptoms in a leaf of M. R. 45 cantaloupes.

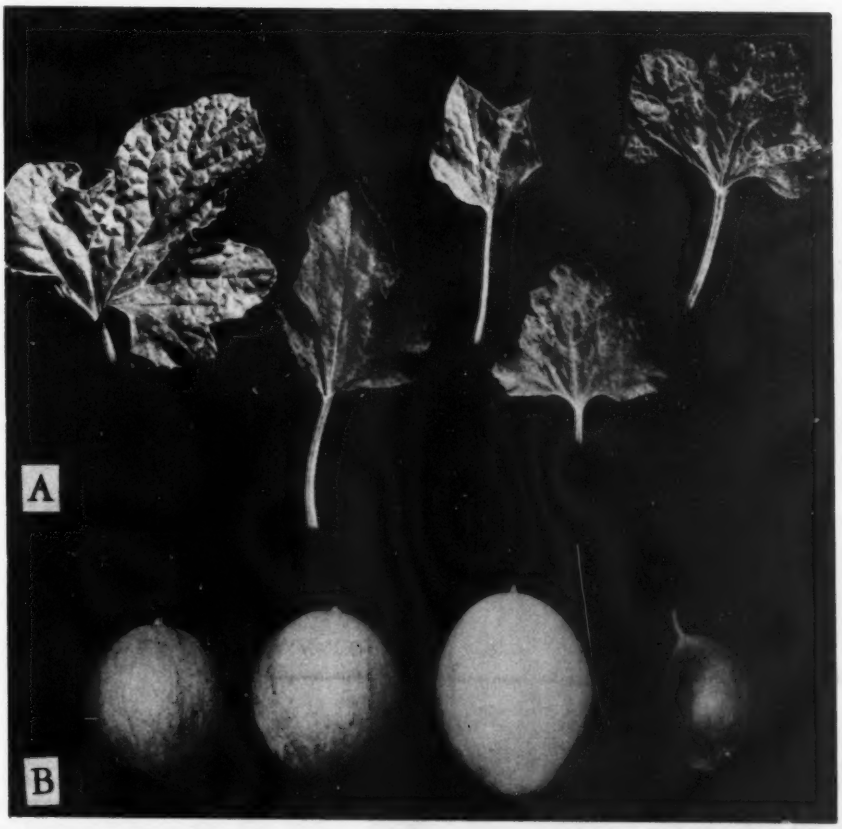

FIGURE 2. Symptoms of yellow mottle virus in leaves and fruits of honey dew. A. At left, normal leaf. Four leaves at right of abnormal shape. Leaf at extreme right has "rugose" surface with enlarged, coarse veins. B. Normal fruit second from right. Other fruits deformed and unmarketable.

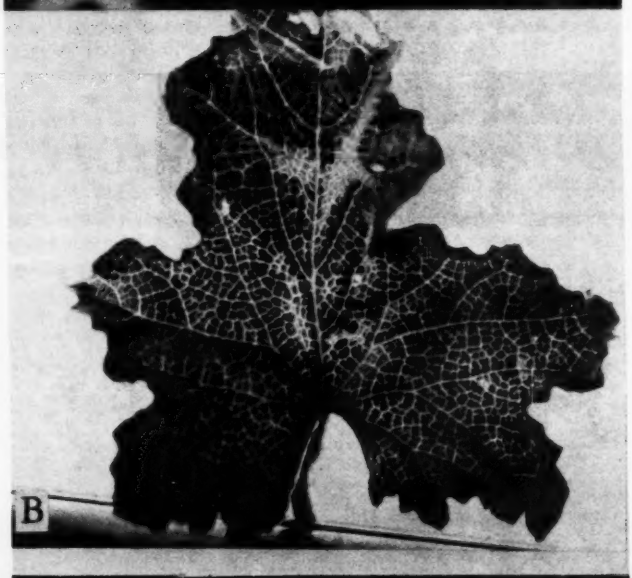

FIGURE 3. Virus-like symptoms in M. R. 45 cantaloupe. A. Dark-green vein-banding symptoms with yellowed interveinal areas. Symptoms of this type are rare in most plantings. B. Yellow mottle and yellow-vein symptoms believed to be associated with the commonly present yellow mottle virus.

FIGURE 4. Aucuba mosaic-like "sectoring" in cantaloupe leaves. At lower left, apparently normal leaf.

Table 2. Incidence of seed-borne mosaics in cantaloupes and honey dew melons at time of emergence in the field, 1949-1951.

Year	Percentages of infected seedlings	
	Cantaloupe	Honey dew
1949	0.8	1.6
1950	0.8	2.2
1951	0.6	1.6[a]

[a] At maturity, 100 percent of the vines were infected, although 2-3 large fruits were set on each plant.

alfalfa side, the percentage of infected plants averaged 48 (ranging from 17 to 90) two weeks previous to harvest. On the sides adjacent to the highways (along which the weeds had been eliminated to a great extent), the incidence of mosaic averaged 23 percent (ranging from 6.5 to 35). The latter figure prevailed in plants near the ranch house, around which many ornamentals were growing.

Similarly, the proximity of aphid-breeding areas to cantaloupes appears to have an influence on the amount, rapidity of spread, and distribution of mosaic. In an 80-acre planting in the Salt River Valley (located just south of Deer Valley), plants of Imperial 45 (M. R.) cantaloupes adjacent to sugar-beets exhibited mosaic symptoms in an average of 45 percent (ranging from 39 to 51). An adjacent 40-acre stand alongside non-developed land which was comparatively free of weeds, showed an average of 2 percent of the vines affected (ranging from 0.5 to 3.5 percent).

Certain virus-like symptoms suggestive of infections by other mosaic or multiple viruses were observed in the field during the past surveys. One of these, a vein-banding type (Fig. 3, A), consisted of dark-green islands of slightly raised tissue along the veins, with brilliant yellow interveinal areas. The percentages of plants showing these symptoms were low in a 40-acre stand in the Salt River Valley. Figure 3B illustrates a yellow-vein type of infection, probably associated with the yellow-mottle mosaic previously mentioned. Brilliant yellow and yellow-green variegations, strongly suggestive of tobacco aucuba mosaic virus infections (Fig. 4), were observed in numerous plants in a 40-acre planting of cantaloupes in the west-central portion of the Salt River Valley, in 1950-51. The exact status of these apparent disorders is not as yet known. Symptoms of curly-top were also noted in cantaloupes, honey dew melons, and watermelons as in previous seasons.

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INTERNAL CORK OF SWEETPOTATOES
PRODUCTION OF CORKY LESIONS IN CORE-GRAFTED ROOTS

L. W. Nielsen[1]

Diagnosis of internal cork, a virus disease of sweetpotatoes, is based upon the presence of necrotic lesions in the fleshy root (3). At harvest many affected roots may not contain these lesions (3, 5), and it is impossible to distinguish healthy roots from those containing the virus. Storing affected roots at warm temperatures for several months will accelerate the development of the lesions (2), and makes it possible to obtain better disease readings. However, 10 to 12 months are required to obtain reliable readings from roots produced in field or greenhouse experiments. This long interval of time between inoculation and disease reading impedes progress in studying the disease.

During the summer of 1950 an observation suggested a procedure for inducing corky lesion production in roots that might reduce the time interval between various types of treatments and disease reading. Cork-free Porto Rico roots grown in California in 1949 were used for plant production in the greenhouse. Ten of the roots were potted individually and produced sprouts in an insect cage covered with a 32-mesh plastic screen. Twenty-five additional roots were similarly bedded and produced sprouts in the open greenhouse several feet from diseased Porto Rico plants. These two groups of roots were bedded in April. The respective pots were emptied in mid-August and the roots examined for the presence of internal cork lesions. Nine of the 25 roots in the open greenhouse had internal lesions believed to be internal cork. None of the ten roots from the insect cage had these symptoms. This observation suggested that the virus was transmitted to the young plants by a vector or other means, and that it was translocated to the mother roots where lesions developed. It further suggested that lesions would develop in healthy Porto Rico roots inoculated by core-grafting with diseased tissue.

In a preliminary experiment started March 30, 1951, internal cork-free roots (1950 California crop) about 1 1/2 inches in diameter were cut in half lengthwise. One-half of each root was core-grafted with corky tissue by the method adopted by Nusbaum (4) and the remaining half held as control. The treated roots were held in a metal moist chamber at room temperature. No lesions were found in control or inoculated half-roots examined one and two months after inoculation, but they were present in inoculated half-roots examined September 6, 1951.

A more extensive test was initiated July 17, 1951 in which whole roots of the disease-free California Porto Rico stock were inoculated with corky tissue. The inoculated roots were bedded in the greenhouse in a sand bed. This section of the greenhouse was maintained at approximately 85°F during the fall and winter months. Twenty-five additional healthy roots were core-grafted with healthy tissue to serve as controls. The control roots were bedded in a flat of soil and placed in a cage screened against insects (32-mesh plastic cloth) in an adjoining section of greenhouse. This section was maintained at approximately 65°F after mid-October when the outside temperatures fell to this level. The inoculated roots were sampled periodically until December 12, 1951 when the remaining roots were examined for cork lesions. The data are summarized in Table 1.

Table 1. The development of internal cork lesions in Porto Rico sweetpotato roots inoculated by core-grafting with diseased tissue July 17, 1951.

Date of sample	Number of roots in sample: Total	Graft alive	Roots with internal cork lesions	Percent roots corky
8-27	29	15	5	17.2
9-18	50	39	9	18.0
10-29	24	17	12	50.0
12-14	12	9	8	66.6
Control*	25	23	0	0.0

* Roots cut 12-20-51

[1]Plant Pathology, North Carolina State College, Raleigh, North Carolina. The assistance of Mr. A. S. Williams in carrying out the work is acknowledged.

The proportion of inoculated roots that developed internal cork lesions increased over the five-month period of the test. The lesions in those roots cut August 27 were 1/16 inch or less in diameter. Small lesions such as these sometimes leave doubt as to whether they are symptoms of internal cork. As the interval between inoculation and sampling increased the lesions increased in size and number. For the last two sampling dates some lesions were 1/2 inch in diameter and the number per root varied from 1 to 6. In all but two cases the lesions were located in excess of 1/8 inch from the margin of the graft. In some cases the lesions developed 2 to 3 inches from the graft.

This method of obtaining cork readings was applied to a number of sweetpotato selections whose roots produced atypical lesions that were light brown or gray in color and with diffuse or indistinct margins, or a few typical lesions. In the early spring of 1950 roots of the selections were inoculated with corky tissue to appraise their reaction to the disease. In all cases the grafts were alive when the roots were bedded. Those selections having the foliage symptoms described by Nusbaum (4) were noted during growth. The roots harvested from the sprouts of inoculated roots were stored at approximately 85°F until April, 1951 when they were examined. Tissue from ten roots of those selections having atypical or a few typical corky lesions was core-grafted into disease-free California roots. Owing to a shortage of insect screened greenhouse space the grafted Porto Rico roots were not bedded until mid-July. The surviving roots grew in flats of soil until December 19, 1951 when they were examined for cork lesions. The data are summarized in Table 2.

Table 2. Development of internal cork lesions in Porto Rico roots core-grafted with tissue from various selections whose roots exhibited few or atypical lesions.

Sweetpotato selection		Foliage Symptoms	Roots of selections having cork lesions	Lesions in Porto Rico roots -- 12-19-51		
Number	Source	7-17-50	April, 1951	No. roots	Grafts alive*	Roots with lesions
			%			
L-21	La.	Yes	8.6	4	2	1
276	S. C.	Yes	13.5	3	1	2
262-27	S. C.	Yes	4.9 ?[a]	2	1	2
HM 18	Miss.	Yes	31.1 ?	10	0	7
L-240	La.	Yes	11.6 ?	6	0	0
HM 15	Miss.	No	29.4 ?	10	2	0

* Grafts alive at time of cutting 12-19-51.
[a] ? Means lesions atypical for internal cork.

Internal cork lesions developed in Porto Rico roots inoculated with root tissue from 4 of the 6 selections. A relatively small number of the grafts survived until the date of examination; however, it is evident that the virus entered the Porto Rico roots prior to the death of the grafted tissue, i. e., in selections 262-27, HM 18, and 276. The data obtained suggest that this procedure can be useful in evaluating the disease reaction of various selections that fail to produce typical internal cork lesions. For selections L-240 and HM 15 the questionable lesions observed in the roots may be related to the causative virus, as the Porto Rico roots inoculated with root tissue from them did not develop cork lesions.

The varieties and selections studied thus far have given three types of disease reaction. Some develop typical internal cork root lesions as exemplified by the Porto Rico variety. The roots of others carry the causative agent, but produce a relatively few, indefinite lesions (selection 262-27), i. e. are symptomless carriers; and others appear to be resistant to the cork producing virus (L-240 and HM 15). If the foliage symptoms are considered in conjunction with the root symptoms, the selections also differ. L-240 and HM 15 are selections of particular interest. At the time of field observation, L-240 had the chlorotic spots and vein clearing with some red pigmentation. However, the cork producing virus was not recovered from the roots. HM 15 had no apparent foliage symptoms, nor was the cork virus recovered from the roots. The performance of these two selections agrees with Martin's observation (1) that the foliage symptoms and

root symptoms may be caused by different causative agents. L-240 is possibly resistant to that agent associated with the cork symptom, and HM 15 is possibly resistant to the cork inducing agent and that associated with foliage symptoms. These preliminary data and observations on varietal reaction to internal cork must be confirmed before more than tentative conclusions are drawn.

Root rotting pathogens as Rhizopus nigricans, Sclerotium bataticola, and Sclerotium rolfsii may destroy many core-grafted roots if they are held some time before bedding, or if they are bedded in an infested medium. Best survival of core grafts and inoculated roots has been obtained when the grafted roots are bedded immediately in moist, warm soil. Disinfesting the bedding soil would probably be a worthwhile precaution. The inoculated roots should sprout and grow in an area protected from insects and at temperatures warm enough (80° to 85°F) for rapid cork lesion development.

The development of cork lesions in Porto Rico roots core-grafted with diseased tissue may be a helpful tool in studying this disease of sweetpotatoes. The procedure reduces the time required for obtaining disease readings, and the tedious process of growing a root crop to demonstrate the effect of various treatments. The method may prove to be an essential step in appraising internal cork resistance in various sweetpotato breeding stocks and selections.

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NORTH CAROLINA STATE COLLEGE, RALEIGH, NORTH CAROLINA

CONTROL OF BACTERIAL HALO-BLIGHT DISEASE OF GARDEN BEANS IN MONTANA[1]

M. M. Afanasiev, H. E. Morris, and H. N. Metcalf

Garden beans are an important field and garden crop in Montana. Practically every farm family and many city inhabitants raise this type of beans for home use. There is also a limited commercial seed production. In general, growing conditions in Montana are very good for the production of high-quality garden beans. However, the occurrence of bacterial blight caused by Pseudomonas phaseolicola (Burk.) Dows. frequently ruins an excellent crop.

In controlling this disease it is usually recommended to use disease-free seed and crop rotation. In preventing secondary spread it is advisable not to work with the beans when they are wet and not to irrigate by the sprinkler method. At present there is very little information available regarding the possibility of controlling spread in the field by different fungicides.

During the growing season of 1951, experiments were conducted at Bozeman, Montana, in which Bordeaux mixture, wettable sulfur dust, and Fermate were tested for field control.

For these experiments ground which had grown no bean crop for a number of years previously was planted with the Top Crop variety, raised in Idaho and free from halo-blight infection. Bean seeds of unknown variety infected with halo blight used as a source of infection were supplied by Dr. W. J. Zaumeyer of the United States Department of Agriculture, Beltsville, Maryland.

Each small plot of beans planted May 28 with the Top Crop variety consisted of four rows of beans 16 feet long. One row of infected beans was planted adjacent to each small plot. A border row of healthy Top Crop beans was also planted. All bean rows were spaced two feet apart.

Each spray material was applied on three randomized and replicated plots. Three check plots were also used. The spray materials were used on the basis of 250 gallons per acre. On this basis each small four-row plot of beans received an application of 0.75 gallons of the spray per application. For this amount of spray 71 grams (a powdered form) of Bordeaux mixture (4-4-50), 21 grams of sulfur (90% of active ingredients), and 5.3 grams Fermate (70% ferric dimethyl-dithiocarbamate) were used. Four drops of DuPont spreader-sticker were used per 0.75 gallons of spray. A three-gallon hand sprayer was used for all sprayings.

The spraying of the experimental plots of beans began as soon as the beans developed the first two leaves (June 23) and continued at approximately weekly intervals until August 17. A total of eight sprays were applied. During approximately the same period (June 15 to August 10), these beans were watered nine times by sprinkling. Chemicals were practically always applied soon after watering.

The rows planted with infected seed were not sprayed with chemicals but were inoculated by spraying with a suspension of Pseudomonas phaseolicola on July 23, 1951.

The first sign of blight infection appeared in inoculated rows on August 3 and in the experimental plots on August 9, 1951. Detailed disease readings were taken on August 16 and August 27. On August 23 severe hail damage occurred in this bean field and approximately 50 percent of the bean leaves were destroyed. Pods from two central rows of each plot were harvested August 16, 21, and 28. Weights of pods from all treatments and replications were recorded.

Table 1. Amount of disease and yield of Top Crop beans in tests for control of bacterial halo blight, Bozeman, Montana, 1951

Treatments	Degree of infection			Yield of bean pods in ounces			
	Leaves	Stems	Pods	Repl. 1	Repl. 2	Repl. 3	Total
1. Check	Medium	None	Slight	333	299	302	934
2. Bordeaux	None	None	None	261	279	289	829
3. Sulfur	Slight to medium	None	Trace	315	322	296	933
4. Fermate	Medium to severe	Trace	Slight	311	215	229	755
5. Inoculated rows	Medium to severe	Trace	Slight	---	---	---	---

[1]Contribution from Montana State College, Botany and Bacteriology and Horticulture Departments, Agricultural Experiment Station, Paper No. 264 Journal Series.

Results (Table 1) show that Bordeaux mixture produced complete control, whereas Fermate had no controlling effect. Beans sprayed with Fermate had the same amount of disease as inoculated rows and more than the unsprayed check. Sulfur gave only a slight beneficial effect in control. The effect of Bordeaux was very outstanding, so that even after severe hail damage this fungicide continued to protect the beans from infection.

Unsprayed beans and plots sprayed with sulfur produced the greatest yield of green pods, and those sprayed with Fermate the lowest. No significant difference in yield was obtained. Spotting and disfiguring of pods with bacterial blight in the Fermate and sulfur plots and in the check plots greatly decreased marketable quality.

It is planned to continue these experiments in 1952.

MONTANA STATE COLLEGE AGRICULTURAL EXPERIMENT STATION, BOZEMAN, MONTANA

THE PREVALENCE OF RING SPOT VIRUS IN A LATENT CONDITION IN CHERRIES[1]

J. A. Milbrath

The stone fruit virus described by Cochran and Hutchins as ring spot (1) is now generally recognized as a common contaminate in many of the stone fruit trees found in commercial plantings. Recent work in Oregon has demonstrated the prevalence of this virus in most of our cherry stocks. In searching for virus-free trees to be used as mother trees for scion orchards for nursery propagation stock, more than 2000 cherry trees have been examined for visible virus and 572 of those which showed no evidence of virus have been indexed (3) for the latent types of ring-spot virus. Many, if not all, of the trees not indexed could be assumed to be carrying the ring-spot virus, but only those trees indexed on flowering cherry will be included in this report.

In selecting trees for indexing on flowering cherry, only those with dark green foliage showing no virus like mottle or break in the normal green pattern were considered as worthy of indexing. Two scion sticks were selected from opposite sides of such trees and a bud from each was placed in Shirofugen flowering cherry. If either bud reacted no further tests were made, but if both buds gave a negative reaction five budsticks were taken from various parts of the tree and again tested on Shirofugen. If these buds proved to be negative the tree was then indexed on Kwanzan, and a negative reaction also on this host indicated the tree to be free from known strains of ring-spot virus. Table 1 summarizes the data obtained during these studies.

Table 1. Cherry varieties from Oregon tested for ring-spot virus.

Variety	No. tested	No. with ring spot	No. without ring spot	Percent infected
Bing	89	74	15	83.0
Royal Anne	122	121	1	99.1
Lambert	53	53	0	100.0
Black Republican	62	62	0	100.0
Black Tartarian	18	18	0	100.0
Montmorency	130	84	46	64.6
Early Richmond	30	30	0	100.0
Late Duke	10	0	10	0.0
Olivet	7	7	0	100.0
May Duke	10	0	10	0.0
English Morello	8	8	0	100.0
Windsor	1	0	1	0.0
Schmidt's Bigarreau	2	0	2	0.0
Deacon	10	10	0	100.0
Centennial	6	6	0	100.0
Waterhouse	3	3	0	100.0
Hoskins	10	8	2	80.0
Black Giant	1	0	1	0.0
TOTAL	572	484	88	84.6

The two varieties of flowering cherry were used because some mild strains of ring-spot virus do not give the necrotic local lesion reaction on Shirofugen, but do cause a severe curling of Kwanzan foliage. A number of Napoleon (Royal Anne) trees appeared to be virus-free when tested on Shirofugen, but gave a strong reaction on Kwanzan. Of the 122 Napoleon trees indexed only one

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tree failed to react on either Shirofugen or Kwanzan. Ring spot-free Bings were found quite frequently, but all Lamberts tested react on both varieties of flowering cherry. Black Tartarian and Black Republican all reacted on either Shirofugen or Kwanzan or on both, but it was not unusual to find a tree that would react only on one or the other. Many of the Montmorency trees used in these studies have come from a nursery which established a mother block for scion wood that happened to have some virus-free trees. Therefore, nearly one-third of the trees indexed proved to be virus-free. If a random sample was taken from the average commercial planting of sour cherries the number of virus-free trees would be relatively few. Likewise, if a sample of sweet cherries were taken with no regard to selection of apparent virus-free types, probably not over 1 or 2 percent of the trees would index virus-free.

The Horticultural Department of Oregon State College, under the supervision of Q. B. Zielinski, has been collecting cherry varieties originating from many different states or countries. These will be tested for resistance to little cherry and albino virus diseases in an attempt to find or develop a variety resistant to these two economically important diseases. They were indexed on Shirofugen for ring spot and also partially screened for other serious viruses by grafting scionwood on the Bing variety. Over 300 varieties of sweet and sour cherry have now been collected, the majority from the U. S. Plant Introduction Garden at Chico, California. Other varieties have come from New York, California, and Canada. The results of indexing these varieties for ring-spot virus are given in Table 2.

Table 2. Cherry varieties from various countries tested for ring-spot virus.

Source	Origin	No. tested	No. with ring spot	No. clean	Percent with ring spot
Chico	Italy	20	7	13	35.0
"	Germany	73	23	50	31.5
"	Australia	8	6	2	75.0
"	Poland	7	2	5	28.5
"	Morocco	4	3	1	75.0
"	England	23	4	19	17.4
"	Mexico	1	0	1	0.0
"	France	34	9	25	27.6
"	Ecuador	1	0	1	0.0
"	USSR	2	0	2	0.0
"	China	1	0	1	0.0
"	India	2	0	2	0.0
"	Europe	5	1	4	20.0
"	Belgium	5	0	5	0.0
"	Switzerland	13	5	8	38.4
"	Unknown	1	0	1	0.0
Canada	---	44	22	22	50.0
California	---	28	25	3	89.4
New York	---	18	11	7	61.1
SUMMARY		290	118	72	40.7

These studies indicate world wide distribution of the ring-spot virus. If these varieties had been indexed when they were first imported the data would have been much more significant. Ring spot commonly occurs in mazzard seedlings as a contaminate from seed transmission (2), and since these introductions have been propagated one or more times since their importation some or all of the ring spot now present could have been introduced in this manner. The stock has also been exposed for several years to natural spread of the virus. The data does show that many of the varieties were virus-free when they were imported and the percentage of ring spot in this stock is still much lower than our native stocks.

These studies show the importance of Horticulturists using virus free rootstocks when developing new varieties.

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OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

THE PEACH LEAF CURL EPIDEMIC OF 1951

H. H. Foster and D. H. Petersen

A leaf curl epidemic, the first since the early 1940's, was observed in the peach areas of northern South Carolina during the 1951 season. Most of the orchards of the Upper Piedmont showed foliage symptoms of leaf curl, (Taphrina deformans). During May some of the severely affected orchards, mostly of the Elberta variety, were nearly defoliated. In these orchards the fruit drop was extremely heavy and frequently insufficient peaches remained to justify continuing the spray program. In orchards showing leaf curl symptoms young infected twigs were often observed but relatively few infected peach fruits were seen.

Certain factors favored the development of this epidemic. Cool, wet weather during late March and early April favored spread and development. This period was preceded by unseasonally warm weather, and it is believed that bud development had begun before the recommended dormant spray of Bordeaux mixture or liquid lime sulfur was applied. However, in many orchards no dormant spray was used. Another contributing factor may have been the lack of a sulfur spray program in 1950 as a result of a peach crop failure. Additional amounts of a readily available nitrogen fertilizer, when applied during the early spring, appeared to be effective in forcing new foliage growth on trees severely affected by the leaf curl fungus.

Table 1. Leaf curl rating for 73 peach varieties.[a]

Variety or Number	Rating	Variety or Number	Rating	Variety or Number	Rating
AGE OF TREES -- 5-6 YEARS					
Early Triogem	3	Starking Delicious	2	Halehaven	1-2
Early Halehaven	3	Sullivan Early Elberta	3-5	Redhaven	0-1
Newday	1	Golden Beauty	1	Southland*.	2
Early Red Fre*	3	Dixired	2	Fort Valley 993	3
Rosebud	2	Dixigem	0-1	Ambergem	1-2
Candoka	2	Southern Glow	1	Missouri	1
J. H. Hale	1-2	Golden Globe	2	Loring	1
Sunday Elberta	2-3	False Sunhigh	2	Ozark	1
Redskin*	2-4	Sunhigh	2	Prairie Rose	2
Summer Crest*	1-2	July Elberta	3-4	Prairie Dawn	0-?
Early Vedette*	1	Early Elberta (Gleason)	1-3	Prairie Rambler	2-3
Fort Valley 110	2	Burbank Elberta	2-3	Davidson Red Leaf	1
Nectar	1-2	Goldeneast	4	Halegold	3-4
Best May	2	Belle of Georgia	3-4	Fireglow	3
Raritan Rose*	1-2	Elberta	4-5	Fairhaven	1-2
Fisher	2	Gemmer's Late Elberta	4-5	South Haven 20	1
Early Fair Beauty	2	Fertile Hale	3-4	Redcrest	1
Triogem*	4	Lizzie	4	Halberta	1-2
Garden State Nectarine	4			Afterglow	4
				Rio Oso Gem	4-5
AGE OF TREES MOSTLY 2-3 YEARS					
Indian Blood (Cling)	1	Fireglow	3	Jerseyland	2
Southland*	3-4	Fairhaven	1-2	Early Jubilee	1
Fay Elberta	1	South Haven 20	1	Cherryred	1
Summer Crest*	1	Redcrest	1	Desirable	1
Fort Valley 20	1	Merrill Gem	2	Halberta	1-2
Early Vedette*	1	Merrill June	1	Redskin*	2-4
Triogem*	1	Raritan Rose*	0-?	Afterglow	4
		Early Red Fre*	1	Rio Oso Gem	4-5

* Variety occurring in both age groups of trees.
[a] Rating = 0 - 5, inclusive.

Table 1 lists the reaction of 73 varieties. The rating used to indicate symptom expression was based on a numerical scale of 0 to 5 in ascending susceptibility. In some peach varieties trees two to three years old showed less pronounced foliage symptoms than trees five to six years in age, but in other varieties foliage symptoms appeared to be similar for trees in both age groups. No variety observed was immune.

BOTANY DEPARTMENT SOUTH CAROLINA EXPERIMENT STATION, CLEMSON COLLEGE, AND BUREAU OF PLANT INDSUTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE, CLEMSON, SOUTH CAROLINA

NUT DISEASES IN OREGON IN 1951

P. W. Miller

Walnut Diseases

Walnut blight (Xanthomonas juglandis (Pierce) Dowson). Walnut blight was widely distributed in Oregon in 1951, causing slightly greater loss than in 1950. Losses from this disease in non-sprayed orchards varied, according to the locality, from a negligible amount up to 30 percent. It is estimated that for the State as a whole, about 5 percent of the potential walnut crop was destroyed by this disease.

Mushroom root rot (Armillaria mellea Fr.). This disease caused the death of a few seedling Persian walnut trees in western Oregon in 1951.

Leaf scorch (non-parasitic). This leaf disorder was widely distributed in Oregon in 1951, occurring to a greater or less extent in most Persian walnut orchards in Oregon. In orchards on soil types very deficient in boron it was exceedingly prevalent, causing a significant decrease in the functional leaf area through death of the tissues and through premature defoliation.

Black-line (girdle) of grafted walnuts (non-parasitic). This disorder was responsible for the death of more grafted Franquette walnut trees in 1951 in Oregon than any other causes. The importance of this disorder in this State was shown by the results of tree by tree survey made in 1951 in 23 widely scattered walnut orchards in which a total of 8507 trees were examined. Out of these 8507 trees, 6405 were of the Franquette variety grafted on Northern California black walnut roots or its hybrid, and 1403 or 21.9 percent were in a declining condition. It was found that 69 percent or 968 of these declining trees had black-line or graft union failure. Not a single case of black-line was found where Persian root stocks had been used. Furthermore, it was found that the incidence of decline due to root and/or collar rots was no greater in the case of Persian root stocks than with the Northern California black walnut root stocks.

Crown rot (cause undetermined). A crown rot of undetermined cause was found affecting a limited number of Persian walnuts grafted on Hinds' black walnut rootstocks in Western Oregon. Most of the affected trees were located in hill orchards on "heavy" soil types.

Filbert Diseases

Bacterial blight (Xanthomonas corylina (P. W. Miller et al.) Dowson). This disease was widely distributed in 1951, occurring to a greater or less extent in many filbert orchards in western Oregon. It was most prevalent in young orchards from one to three years of age, causing the death of a considerable number of young trees. It also occurred in many older orchards where it caused the death of many buds and twigs.

Mildew (Phyllactinia corylea (Karst.) This disease occurred in greater or less amounts in many orchards in western Oregon in 1951 but was of no economic importance as it did not appear until very late in the season. The infected leaves retained their normal green color until the end of the season and persisted on the trees until normal leaf fall.

UNITED STATES DEPARTMENT OF AGRICULTURE, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, DIVISION OF FRUIT AND NUT CROPS AND DISEASES, OREGON STATE COLLEGE, CORVALLIS, OREGON

A HITHERTO UNREPORTED CORN DISEASE[1]

W. E. McKeen[2]

On July 24, 1950, a hitherto unreported corn disease was observed on a few of the plants in a segregating inbred in the corn plots on the Experimental Station, Harrow, Ontario. The affected strain of corn had been obtained from Guatemala five years previously, and no malady had been detected on it in the intervening period. Numerous isolations from incipient lesions yielded only Curvularia inaequalis and an Alternaria sp., neither of which proved to be pathogenic.

By the latter part of August all of the infected plants, 15 in number, had died without producing seed. However, seed was obtained from plants of the same variety with a similar history and was planted on May 15, 1951. The disease first appeared on the new plants on June 27, when they were about three feet high (Fig. 1), with slightly less than 1 percent of them showing symptoms. Some of these plants were dusted with copper oxychloride sulfate, but unfortunately it proved toxic and both the dusted and undusted plants succumbed. Again, Curvularia inaequalis and an Alternaria sp., were isolated.

The necrotic lesions begin as tiny dots and relatively large alternately light and dark rings form around these points until the lesions are up to one inch in diameter (Fig. 2). These zonate lesions always appear on the tip of the oldest leaf first, as is typical of Helminthosporium leaf spot, but they rapidly spread back to the sheath and the whole leaf withers and dies. After the outer third of this leaf is covered with lesions (Fig. 3), lesions begin to appear on the next oldest leaf, and so on up to the stem. Each leaf soon dies and within a few weeks the whole plant is dead, usually before the youngest leaf is attacked.

Whether this disease is caused by a microbe or is of genetic origin is not known, but apparently it occurs only when certain gene combinations are present in the plant. Although C. inaequalis has not proved to be pathogenic on leaves under any of the conditions used in the present studies, its pathogenic capabilities must not be ruled out until it is tested on susceptible plants. It may be mentioned that Groves and Skolko (1) reported C. inaequalis on corn seed in 1945.

This disorder is only of academic interest since it is fatal to all plants of a certain genotype, and thus probably will never become widespread.

Literature Cited

1. Groves, J. W. and A. J. Skolko. Notes on seed-borne fungi III. Can. J. Res. 23: 94-104. 1945.

DOMINION LABORATORY OF PLANT PATHOLOGY, HARROW, ONTARIO

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[1] Contribution No. 1150 from the Division of Botany and Plant Pathology, Science Service, Department of Agriculture, Ottawa, Canada.

[2] Associate Plant Pathologist, Dominion Laboratory of Plant Pathology, Harrow, Ontario.

(See page 143)

FIGURE 1. A young corn plant of Guatemala ancestry, which is suffering from the leaf spot. Observe that the oldest leaves have been killed and that infection begins at the tips of the leaves. The youngest leaves are healthy.

FIGURE 2. A section of a leaf showing large zonate lesions.

FIGURE 3. Three leaves showing various stages of disease: the bottom leaf has a few lesions near its tip; the middle leaf has more lesions, and the upper leaf is almost dead.

A SEED-BORNE DISEASE OF CORN[1]

W. E. McKeen[2]

In July, 1949, a spotting was observed on one of the corn inbreds (#13) at Harrow, Ontario, after three years of selfing, but this spotting was not present on a sister inbred which differed only in that its seed originated from another selfed plant in the second generation. Since no organism was observed in the young lesion and since no parasitic fungus or bacterium could be isolated, it appeared that this disease was either of virus or physiological origin.

The diseased plants showed a wide range of symptoms, and several of the severely diseased ones did not develop ears (Fig. 1). Some plants had only a few lesions on each leaf (Fig. 5); others were completely covered with rings (Fig. 4), except a few of the lower leaves. Lesions frequently occurred on the husks (Fig. 2) and sheaths. A minute clear spot, frequently with a necrotic centre, was the first indication of this disease and, later, alternatively clear and necrotic concentric rings (Figs. 3 and 4) formed about this spot and frequently the lesion became an inch, or occasionally two inches, in diameter and coalesced with other spots to give the lesion an irregular appearance. These large lesions tended to be oval in shape, the long axis being parallel to the veins. When numerous tiny lesions were extremely close together, a mottle resulted and no concentric rings formed.

Environment influenced the symptom expression to a considerable degree. Disease expression was most intense during bright hot weather. During the cool dark winter months, the lesions were always minute and faint and frequently the plants remained in an apparently normal condition.

This disorder has proved to be seed-borne. Numerous attempts to transmit this disease by mechanical inoculation to Nicotiana tabacum, millet, sorghum, oats, wheat, barley, and several corn inbreds and hybrids have not been successful. Grafting was of no value since grafts did not "take". Aphis maidis did not transmit this disease.

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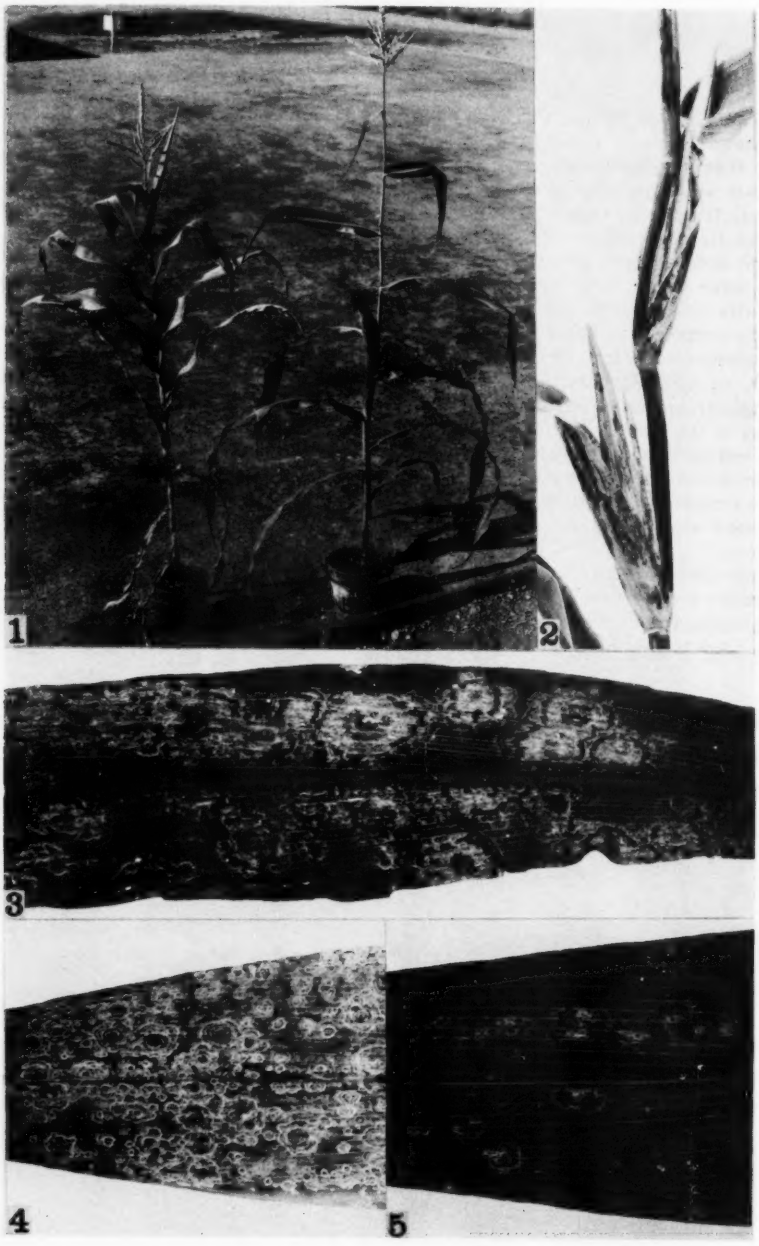

(See page 145)

FIGURE 1. Two corn plants: the one on the right is healthy, the one on the left is diseas and its lower leaves have died.

FIGURE 2. Corn cobs, with numerous lesions on their husks.

FIGURE 3. A section of a diseased corn leaf bearing several large lesions.

FIGURE 4. A portion of a leaf with numerous concentrically ringed lesions.

FIGURE 5. A few ringed lesions scattered over a section of a corn leaf.

MEXICAN VARIETIES OF WHEAT RESISTANT TO RACE 15B OF STEM RUST

N. E. Borlaug[1], A. Campos T.[2], and B. B. Bayles[3]

Race 15B of stem rust (Puccinia graminis var. tritici) of wheat became widely distributed for the first time in North America during the summer of 1950 when it was found throughout most of the central part of the United States and Canada[4]. This race[5] was found in Mexico for the first time during April 1951 in three widely separated geographic areas, namely, the States of Sonora (Northwest), Nuevo León (Northern), and Hidalgo and Mexico (South Central). There was no commercial damage, however, on the winter crop in these areas since the inoculum did not build up until the crop was approaching maturity. Temperatures and precipitation during the winter of 1950-51 were below normal in northern Mexico and probably accounted for the fact that stem rust did not become generally prevalent in the fall-seeded crop until it was nearly ripe. The summer crop, which is planted over a wide geographic area in May and June, was found generally infected. Severe losses occurred in some areas. Race 15B was the most prevalent race in the States of Jalisco, Guanajuato, Michoacán, Querétaro, Hidalgo, Mexico, Puebla, and the Federal District. This was the first time that stem-rust damage occurred on the varieties Supremo and Chapingo, which are resistant to the other races of stem rust prevalent in Mexico. Race 15B was found on commercial plantings of barley and also was widespread on volunteer wheat and barley plants in bean and corn fields in many parts of the central region.

Reaction of Mexican Commercial Varieties to Race 15B

The very severe epidemic in the summer of 1951 gave an excellent opportunity to study the field reaction of commercial varieties to this new race of rust. Two new varieties of wheat, developed by the Oficina de Estudios Especiales of the Secretaria de Agricultura y Ganaderia in collaboration with the Rockefeller Foundation, have been found to posses excellent resistance to 15B and to all other races of stem rust prevalent in Mexico. The varieties Kentana 48 (a cross of Kenya x Mentana) and Lerma 50 (a second backcross of Kentana to Mentana) were entirely free from rust when grown adjacent to fields of Supremo and Chapingo, which were killed. The resistance of Kentana and Lerma was uniform over the entire area wherever they were being grown in commercial plantings. Two other commercial varieties, Kenya Rojo RF-324 and Kenya Blanco RF-321, distributed by the Ministry of Agriculture of Mexico in 1946, were also found to be highly resistant. Kenya Rojo is the parent which has contributed the resistance to 15B to both Kentana and Lerma. Mentana, the other parent of both Kentana and Lerma, has a degree of seedling resistance to race 15B. The two Kenya varieties are still being grown commercially in some areas in Mexico but will be replaced rapidly by Kentana and Lerma because of their higher yields and earliness.

Sufficient seed of Kentana was harvested during October and November 1951, to plant approximately 40,000 acres to this variety during the current winter season. Consequently, by the time the 1951-52 winter crop is harvested, there will be adequate seed available to plant a large percentage of the Mexican wheat acreage to this variety. Lerma 50 is a newer variety which is being grown for the first time by Mexican farmers. Approximately 1,500 bushels of seed were harvested and all of this was planted during December. Kentana yields considerably better from December plantings than from June plantings, whereas Lerma is equally well adapted for winter or summer plantings. Both varieties are of spring habit, and both are susceptible to leaf rust. Kentana is red-grained, whereas the grain of Lerma is white.

The commercial varieties Yaqui, Mayo, Chapingo, and Nazas, carrying Newthatch type of stem rust resistance, were highly susceptible during the past summer. The variety Supremo, which has Surpresa and Hope as its stem-rust resistant parents and is the most extensively cultivated variety in Mexico at the present time, is highly susceptible. All of these varieties have

[1]Rockefeller Foundation, Mexico, D.F., Mexico
[2]Ministry of Agriculture, Mexico, D.F.
[3]U.S. Department of Agriculture, Beltsville, Maryland, U.S.A.

[4]Stakman, E.C. and Loegering, W.Q. Physiologic races of Puccinia graminis in the United States in 1950. U.S. Dept. Agr., Bur. Entom. and Plant Quar., and Bur. Plant Indus., Soils, and Agr. Engin., and Minn. Agr. Expt. Sta. 16 pp. (Processed.) July 1951.

[5]Collections were identified as race 15B by E.C. Stakman and W.Q. Loegering of the Division of Plant Pathology and Botany, University of Minnesota, and Bureau of Entomology and Plant Quarantine, U.S. Dept. of Agr., St. Paul, Minn.

been resistant in the field to all of the races of stem rust found in Mexico with the exception of race 15B.

Reaction of Breeding Materials to 15B Under Mexican Conditions

Four large wheat-breeding nurseries were grown during the summer of 1951 by the Oficina de Estudios Especiales in Mexico. These nurseries were seeded in June in four widely separated areas namely, Chapingo, Mexico; Mexe, Hidalgo; Irapuato, Guanajuato; and Guadalajara, Jalisco. In addition to the breeding material from the Oficina de Estudios Especiales which consisted of approximately 60,000 lines and varieties, a large number of lines, varieties, and collections were evaluated through a cooperative arrangement with the Divisions of Cereal Crops and Diseases and Plant Exploration and Introduction of the U. S. Department of Agriculture. This material included:

1. 1,007 of the most promising lines and standard varieties from the United States and Canadian breeding programs.
2. 4,702 foreign introductions -- (U. S. D. A. World Wheat Collections).
3. 200 miscellaneous introductions and lines from various South American countries.

Severe stem-rust epidemics, due principally to race 15B, developed at all four locations. The epidemics were especially severe at Mexe, Hidalgo, and Chapingo, Mexico, where the susceptible lines were generally killed. The nursery at Mexe, Hidalgo, was in a valley where Supremo was the predominant variety. The build-up of stem rust was so severe that many fields of this variety with a potential yield of 40 bushels were so badly damaged that they were not harvested. The inoculum in the Mexe nursery may, therefore, be assumed to have been largely race 15B. In the nursery at Chapingo, Mexico, inoculum of stem rust races 15B, 17, 19, 38, 56, and 59 was put into the nursery to supplement natural inoculum. The diversity of material planted in these nurseries, where severe epidemics developed with 15B one of the dominant races, made it possible to obtain a large amount of valuable information concerning the reaction of commercial varieties and the effectiveness of different types of resistance which currently is being used in the breeding programs. These field data can be summarized as follows:

1. All of the commercial varieties of spring wheats (bread and durums), currently grown in North America with the exception of Kentana 48, Lerma 50, Kenya Rojo, and Kenya Blanco, were severely rusted. Cadet, although susceptible, was somewhat less so than the other northern hard red spring wheat varieties.
2. Parental varieties or lines that are being used in bread wheat breeding programs in North America and which were found to be resistant or highly resistant included:
   (a) Kenya 58, Kenya 117-A, Kenya 338-AC.2.E.2, Kenya 291-J.I.I.I, Kenya 294B.2.A.3, Kenya 338-AA.1.A.2, Kenya RF-324, Kenya 130.B.6.B RF-321.
   (b) Red Egyptian.
   (c) McMurachy.
   (d) Egypt Na-95.
   (e) No. 43 (South Africa) P. I. 159106.
   (f) McMurachy-Exchange, RL-2265 (Canadian)
3. Parental varieties or lines that are commonly being used in breeding programs and which were found to be susceptible included:
   (a) Hope.
   (b) Egypt Na-101, Kenya Standard, and Kenya Governor[6].
   (c) Fronteira, Surpresa, Bage, Frontana, Rio Negro, Supremo, Redman, Newthatch, and Lee.
4. Bread-wheat introductions from World Wheat Collection (tested for the first time) which were highly resistant:
   (a) Transvaal Africa -- P. I.-170915, P. I.-170916, P. I.-170917, P. I.-170918, P. I.-170919, P. I.-170922.
   (b) Argentina -- H-797-49-4812, P. I.-189812.
   (c) Spain -- E.M.V. #1 P. I.-191426.
   (d) Portugal -- 117-AFS-1456, P. I.-192097.
   (e) Kenya 501 Tipo 231, P. I.-192154.

[6]These varieties probably were killed by a race other than 15B.

(f) Kenya, P. I. -192173.
(g) Kenya, P. I. -192182.
(h) Portugal -- V x 9 Maria 9 D_2 P. I. -192465.
(i) Portugal -- Roci Egipties 472 P. I. -192468.
(j) Portugal -- T. vulgare x Aegilops ovata 39, P. I. -192480.

5. Durums or Poulard wheat (one T. persicum) introductions from World Wheat Collection which were highly resistant:
(a) Canada -- Golden Ball-Iumillo-Mindum, RL-1714.
(b) Portugal -- Tremez Preto, P. I. -56256-3.
(c) Portugal -- Tremez Rijo, P. I. -56257.
(d) Portugal -- Tremez Rijo, P. I. --56257-1.
(e) Portugal -- Tremez Molle, P. I. -56258-1.
(f) Arabia -- P. I. -145720.
(g) Spain -- Almendral P. I. -190990.
(h) Spain -- Amapole P. I. -190993.
(i) Spain -- Caravaca #1 P. I. -191054.
(j) Spain -- Claro de Bazalote P. I. -191076.
(k) Spain -- Recio de Toledo P. I. -191192.
(l) Spain -- Rojal de Alicante P. I. -191193.
(m) Spain -- Rojal de Almeria P. I. -191194.
(n) Spain -- Recio Dan Clemente P. I. -191251.
(o) Italy -- St-464, Ethiopia P. I. -191365.
(p) Portugal -- Anafiel Claro 3291 v86 P. I. -191743.
(q) Portugal -- Carnadi Abdu tipo 103 P. I. -192168.
(r) Portugal -- Amarai blanco tipo 142 P. I. -192179.
(s) Portugal -- Egypto 2100 P. I. -192502.
(t) Italy -- T. persicum fuliginosum P. I. -191395.

6. Crosses and the number of advanced generation lines from different breeding programs in America which showed high degree of resistance.

Cross	Number of lines	Country
Kenya R.F. 324 x Mentana	Several hundred	Mexico
Kenya R.F. 324 x Mentana^2	60	"
Kenya R.F. 324 x Mentana^3	Several hundred	"
Maria Escobar^2 x Newthatch	85	"
(Kenya-Marroqui^2) Maria Escobar	15	"
Egypt Na-101 x Timstein	65	"
(Aguilera-Kenya)x(Marroqui-Supremo)	10	"
Kenya R.F. 324 x Candeal	35	"
Kentana x Yaqui	170	"
Frondoso x Kenya C-9906	9	Texas, U.S.A.
Renacimiento x Kenya C-10862	27	"
Surpresa x Kenya-Gular C-4913	5	"
RL-2265 x Redman	2	Canada
RL-2265 x Redman^2	3	"
RL-2265 x Redman^3	2	"
Thatcher x (RL-2265-Redman^2)	6	"
(Mida-Cadet)x(RL-2265-Redman^2)	1	"
Frontana x (RL-2265-Redman^2)	1	"
Timstein x Kenya 58	1	Minnesota, U.S.A.
Kenya-Gular-Pilot x Kenya 58-Newthatch	2	"
Frontana x Thatcher	15	"
Red Egyptian x Frontana	3	"
Frontana x Kenya 58	5	Brazil, S.A.
Red Egyptian x Rio Negro	2	"
Red Egyptian x Rio Negro^2	5	"
Castelar 49-4131, line G 1-3-3-1	1	Argentina, S.A.

Development and Spread of Race 15B in Mexico

There is considerable circumstantial evidence that the spores which gave rise to the 1951 summer 15B epidemic in Mexico were blown into Mexico from the United States and Canada during the fall and winter of 1950-51. There was relatively little survival of these spores in northern Mexico during the winter season of 1950-51, probably because of the extensive drought and cold weather in that area. The rust overwintered, however, in many areas on the Pacific Coast and in south central Mexico. In the latter area it increased to epidemic proportions during the summer of 1951 and caused severe damage to commercial plantings.

ROCKEFELLER FOUNDATION, MEXICO, D. F., MINISTRY OF AGRICULTURE, MEXICO, D. F., MEXICO; AND U. S. DEPARTMENT OF AGRICULTURE, BELTSVILLE, MARYLAND, U. S. A.

WHITEHEAD OF OATS IN MINNESOTA 1950-51

S. Goto, J. W. Gibler and A. Buzicky[1]

Bleached sterile panicles on otherwise normal appearing oat plants were prevalent in Minnesota during 1950 but not in 1951. The condition was due in part to the destruction of the tender portions of the culm by the common stalk borer, Papaipema nebris, the European corn borer, Pyrausta nubilalis, the wheat stem maggot, Meromyza americana, and in at least two cases by two species of thrips, Anaphothrips obscurus and Chirothrips cuneiceps.

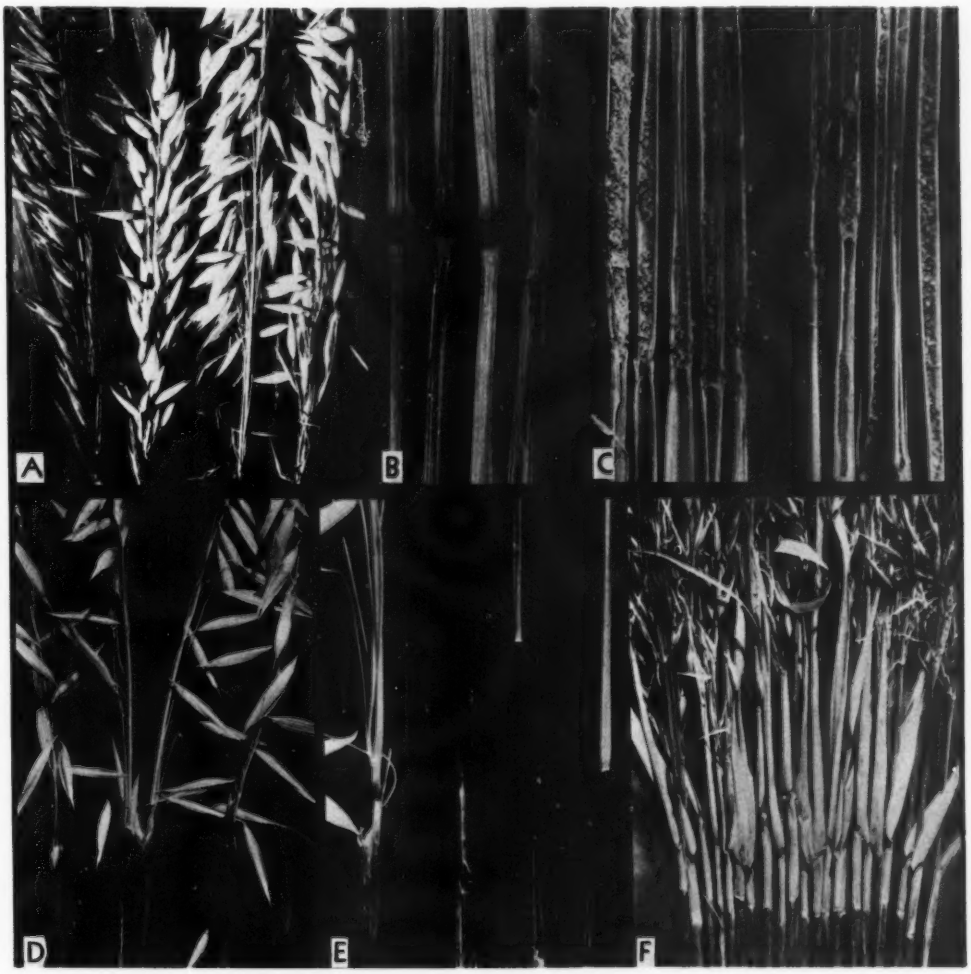

FIGURE 1. A. Three "whiteheads" beside a normal panicle of oats.
B. Entry holes of common stalk borer.
C. Frass filled internodes with common stalk borers at points indicated.
D. "Whiteheads" with cut flag leaf, cause unknown.
E. Cut stems of D.
F. "Leaf drop" showing discoloration above girdle.

[1]Paper No. 2795. Scientific Journal Series Article, Minnesota Agricultural Experiment Station, University Farm, St. Paul 1.

The characteristic symptom is the "whitehead" protruding from the green flag leaf (Fig. 1A). Attack by either of the borers is readily recognized by the entry or exit hole on the stem (Fig. 1B) and the frass packed internode (Fig. 1C). In 1950 the common stalk borer was noted attacking oats in Olmstead, Jackson, Murray, Hennepin, Le Sueur, Dakota and Ramsey Counties, especially near the edges of the fields. The corn borer was found on oats only in the experimental plots at St. Paul. In 1951 there was little borer attack on oats, the "whitehead" symptoms being due primarily to the stem maggot.

In 1950 at University Farm, another "whitehead" of oats was observed, in which the flag leaf had been cut off (Fig. 1D). The culm also appeared to be cleanly cut inside the flag leaf sheath, and the cut ends were separated as much as two inches, possibly due to the basal growth of the enclosing sheath (Fig. 1E). There was no indication of insects or insect frass.

The reverse symptoms, that is, normal panicle with bleached flag leaf, similar to the "leaf drop" reported by A. J. Mix on wheat in Maryland (Plant Dis. Reptr. 29: 583-586. 1945) was found at St. Paul in 1950 but not in 1951. A groove girdled the leaf sheath 1 to 2 cm. below the flag leaf (Fig. 1F). The portion above this girdle was dead and yellow, while the rest of the plant including the panicle remained normal. Heavy winds and rain knocked these leaves down, to give the hanging appearance described by Mix. No trace of the causal agent was found. Damage due to thrips is not figured but superficially resembles Figure 1A.

MINNESOTA AGRICULTURAL EXPERIMENT STATION, UNIVERSITY FARM, ST. PAUL

OAT DISEASES IN ARKANSAS 1950-51

H. R. Rosen

For the sixth successive year, the fall of 1950 was unfavorable for the seeding of winter oats, so that the acreage took a further drop below that of 1944. Up to the third week of September there were abundant rains so that growers who seeded during that period obtained good stands in most instances. October and November, however, were very dry and since most of the seeding is usually done in those months, the dry weather seriously interfered with preparation of seed beds and with planting. The stands obtained were generally thin and often irregular.

While there were some aphid infestations on these fall stands, they were not particularly heavy and by no means so common or abundant as leaf hopper infestations. The latter were so severe that oat fields generally presented a silvery or grayish-green appearance at a distance. Despite a severe winter, leaf hoppers persisted and apparently increased in great abundance during the succeeding spring. Spring-sown oats suffered even heavier infestations than winter oats and the very poor yields obtained from spring oats were due probably in large measure to these infestations. In the varietal test plots maintained jointly with the Agronomy Department at the University Farm, Fayetteville, the five highest yielders among the spring oats, in bushels per acre, were: Missouri 0-200, 38.8; Missouri 0-4201, 38.6; Andrew 38.2; Neosho 35.9; and Columbia 32.9. These compare with the five highest yielding winter oats as follows: Arkwin 63.5; R22-36b-9-647, 60.5; Fulwin 58.9; R22-36b-9-500, 58.4; and Traveler 56.5 bushels per acre. While a difference of 40 percent in yield of winter oats over spring oats has been an average during the last 30-odd years, the difference of over 60 percent obtained in 1951 can best be attributed to differences in leaf hopper infestations.

Crown Rust (Puccinia coronata) and Its Relation to Winter Injury. With abundant rainfall during the summer of 1950, volunteer oats came up in unusual amounts and became severely infected with crown rust. Most of this proved to be biotypes of race 45 or 57 with a sprinkling of the new race 55 or 101 (see P. D. R. 35: 370-371. 1951). As noted in the publication just cited, crown rust apparently spread from these volunteer plants to oats that were sown early in September. With abundant rains that fell shortly after these oats were up, the epidemic of crown rust that developed subsequently was one of the most severe that has been observed in over 30 years. By the end of October fully 80 percent of the leaf area was infected in all susceptible varieties, as noted in the publication cited.

The epidemic was extensive and seemingly occurred throughout the State on all early-planted oats. Even Red Rustproof strains, including Ferguson 922, Nortex, Delta Red, and Nortex 107, were so badly rusted that whole fields looked yellow instead of green. All Bond varieties except one (Arkwin), and all Victoria derivatives suffered severely, although most of the latter were recorded as having 30 percent of the leaf area infected when infection of the former was recorded at 80 percent or more. As noted previously (P. D. R. 35: 149-151. 1951), the Red Rustproofs are not as rust-escaping to the Bond-infecting races as they are to the older races, and this appears to be true for seedlings as well as for older plants under natural conditions.

Since the predominant oat acreage of Arkansas and of the South as a whole consists of Victoria derivatives and Red Rustproofs, this region is seemingly faced with an increasingly serious problem in the growing of oats until new varieties are developed that embody the crown rust resistance of Landhafer or Santa Fe, or are otherwise resistant or escaping to these newer races. The new winter oat variety Arkwin, while partly resistant and escaping to at least some of the biotypes of race 45 or 57, is seemingly fully susceptible to race 55 or 101, and may act as an incubator or spreader of this race, especially if used on large contiguous acreage.

Unlike the early fall-planted oats that became severely infected with crown rust in 1950, late-planted oats escaped infection to a very large degree, mainly because of the dry weather that prevailed from October through December.

The winter of 1950-51 was exceptionally severe with 0°F. or below occurring on one or more days throughout the State. Almost all badly rusted oats failed to survive the winter while those that were relatively free from rust suffered very little or were in part killed, depending on their degree of winter hardiness. Thus, while early-planted Traveler oats were mostly killed during the winter, late-planted Traveler suffered comparatively little. This was in contrast with less hardy varieties such as Victorgrain, Fulgrain, and the Red Rustproofs, which survived rather poorly even where sown late.

It seems that the combination of a heavy fall crown rust epidemic and a severe winter were largely responsible for the relatively low average State yield. Even in fields where stands were not greatly reduced, the plants apparently were so injured by cold weather that they failed to make

normal growth thereafter. However, in most instances the stands were considerably reduced and numerous fields were abandoned or plowed up in the spring, as revealed in the survey made in May and June in 50-odd important oat-growing countries.

This survey, unlike the one in the previous fall, revealed comparatively little crown rust. Despite the severe fall epidemic, the rust was apparently killed with the killing of the host plants, and the slight amount found in the succeeding spring was probably blown in by winds from more southern regions.

Out of 20 collections obtained mostly in May and June, 1951, which still possessed viable spores during the winter of 1951-52, 14 consisted wholly of biotypes of race 45 or 57, while 6 yielded both 45 and 101, with race 45 predominating in 19 of the 20 collections. The only 1951 collection showing race 101 predominating was obtained on October 8 on volunteer oats at the University farm, Fayetteville. This appears to be the first collection obtained by the writer in which this race predominated. (It may be of interest to those engaged in race identification to note that Letoria is a good differential variety to distinguish race 45 or 57 from race 55 or 101.)

Leaf Spot (Pyrenophora (Helminthosporium) avenae). Leaf spot appeared abundantly, together with crown rust, in the fall of 1950 on volunteer oats from which it spread to fall sown oats. However, the amount of leaf spot was considerably less than of crown rust, and while some probably overwintered, spring infections were comparatively few and not nearly so abundant as in the past few years.

Anthracnose (Colletotrichum graminicola). This disease, like leaf spot, was much less common in 1951 than in the past few years, and although it could be found in almost any field and seemingly caused some damage in the extreme southern part of the State especially in Hempstead County, it was unimportant as a whole. Possibly the exceptionally severe winter had something to do with reduction in leaf-spotting types of parasites.

Helminthosporium Blight (H. sativum var.) (H. victoriae). A slight amount of blight was found in the fall at the University farm on early planted Victoria derivatives and to a greater degree in commercial fields of Traveler in Sebastian County, also sown early. Where early fall seeding is practiced in order to obtain good fall and winter grazing, it seems obvious that Victoria derivatives susceptible to blight are unsafe to use. Of the newer Victoria derivatives that have been released recently, including Arlington, Atlantic, Mustang, and Coy, only Coy appears to be resistant (but this is unfortunately tied up with greater crown rust susceptibility).

Smuts (Ustilago avenae and U. kolleri). As a whole there appeared to be less smut than was recorded in the past few years, although a few individual fields were found where as much as 30 percent of the plants were smutted. This included commercial fields of Red Rustproofs as well as of Traveler. Evidently the new races of smut that attack Victoria derivatives are now pretty well distributed in Arkansas.

Red Spot Mosaic. There was a decidedly smaller number of plants exhibiting symptoms of this disease in 1951 than in the past few years. Likewise, there was a much reduced aphid infestation. Although the writer has not succeeded in correlating any possible insect carrier, including aphids, with this malady, the recent work of Oswald and Houston in California (P. D. R. 35: 471-475. 1951) on a virus disease of barley, wheat, and oats, which they found to be transmitted by four species of aphids suggests that aphids may be the carriers of red spot mosaic. In the field they noted a "reddening of oats" and in the greenhouse, by using viruliferous aphids, they obtained "irregular yellowish-green blotches on the older leaves principally near the tips", and "these spots later take on a reddish cast and eventually the leaf tip and often the entire leaf becomes reddish-brown in color; associated with this discoloration is extreme dwarfing" and a "longitudinal chlorotic striping of the new leaves."

These symptoms are in general comparable to those described by the writer for red spot mosaic (P. D. R. 32: 172-175. 1948, and 35: 151. 1951), but the details presented by Oswald and Houston appear to be quite different, especially in the fact that diseased plants in Arkansas very often recover and give good yields while nothing is said of such recovery in California. However, differences in varieties and especially in climatic conditions render such comparisons inadequate, and the findings by Oswald and Houston of absence of soil transmission and inability to produce the disease by mechanical means are directly comparable to those found by the writer in red spot mosaic in Arkansas. Nevertheless until the two diseases are brought together and compared side by side, using comparable varieties or the same varieties, there is no assurance that they are identical.

Stem Rust (Puccinia graminis). As in 1950, there was very little stem rust on oats in 1951, and what there was was confined almost wholly to spring-sown oats.

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AN OUTBREAK OF PHYLLOSTICTA CANKER AND LEAF SPOT OF SOYBEANS IN MARYLAND[1]

R. A. Jehle, Anna E. Jenkins, K. W. Kreitlow, and Helen S. Sherwin[2, 3]

In Maryland, where soybeans (Glycine max (L.) Merr.) have long been an important field crop, 78,000 acres were sown last year, and the 1,232,000 bushels threshed were valued at $3,388,000.[4] Comparatively little attention had been given to diseases of the crop in the State prior to a survey conducted by the University of Maryland (10).

The first discovery in Maryland of the soybean disease commonly known as Phyllosticta leaf spot (6, p. 80) was made under the Emergency Plant Disease Prevention Project (1943-1945). In 1944 it was found in Kent (9, 12), Queen Annes (9, 12, 13), Caroline (9, 13), Dorchester (13), and Worcester (12) Counties on the Eastern Shore, and in Prince Georges County (13) and Carroll County (12) west of Chesapeake Bay. Attack ranged from moderate to severe. During 1945-50 loss from the disease in the State was neligible. In late August and September 1951, however, Phyllosticta stem and pod canker, as well as leaf spot, was abundant in plantings at Salisbury, Wicomico County.

The diseased condition of the soybeans at Salisbury was brought to our attention in the following manner: Pathologists and county agents in Maryland had been alerted concerning the existence of a scab disease of soybean (4). Shortly thereafter, a commercial grower consulted the local county agent regarding the poor stand and severe stunting of soybeans planted in a field that had formerly been a peach orchard. On August 24, F. L. Bentz, Assistant Agronomist of the University, visited the field and obtained specimens of the stunted plants which were brought to the Division of Plant Pathology at the University of Maryland. The specimens immediately attracted attention because of the presence of small, superficial cankers on the stems, simulating those of the scab disease. The cankers were up to 2 mm. wide by 4 mm. long, grey to almost white, with narrow purplish to brown borders. Pod cankers and leaf lesions also were present.

On August 29 the senior writer examined the field and obtained additional specimens for diagnosis. The severely stunted soybeans sown in the former peach orchard were of the variety Hawkeye. Neighboring fields of soybeans consisted of the varieties Hawkeye, Lincoln, Monroe, and an undetermined variety. These several fields, with their acreages and the relative severity of the disease in each, are shown in Figure 1.

Many plants were examined in the two fields of the Hawkeye variety. Both fields had been planted with seed from the same lot. Stem cankers were found on practically every plant and were numerous on some. In field 2, where there was no evidence of stunting, cankers were usually larger (1-4 mm. wide x 1-12 mm. long) and deeper than those on plants of the original sample from field 3. Pod and peduncle cankers were common and were conspicuous. Some of the pod lesions extended to the seed (Fig. 2, A-C). Dark punctiform areas, which proved to be pycnidia of the pathogen, were present on many of the cankers.

Since the disease was equally prevalent in both fields of Hawkeye, it was apparent that some additional unknown factor was involved in the stunting of the crop in the former peach orchard.

Subsequent observations, on September 8, were confined chiefly to field 2. Most of the lower leaves had already fallen. Many of those remaining on the upper part of the plant had brown, necrotic lesions, usually at the margins but sometimes scattered over the entire leaf (Fig. 2, D). In many cases the leaves appeared tattered from shattering of the diseased tissue. Pycnidia were often abundant on these dead areas.

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[1]Scientific Article No. A. 350, Contribution No. 2330, Maryland Agricultural Experiment Station (Department of Botany), University of Maryland. Joint contribution presented at the Ninth Annual Meeting of the American Phytopathological Society, Potomac Division, Beltsville, Md., February 27, 1952.

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[3]The writers wish to express their appreciation to Drs. J. M. Crall, Lee Ling, Lindsay S. Olive, L. R. Tehon, and E. A. Walker for desiderata that have aided in their etiological study of this disease and in the preparatopn of the manuscript.

[4]Information supplied by the Crop reporting Service, Bureau of Agricultural Economics, U. S. Dept. of Agr.

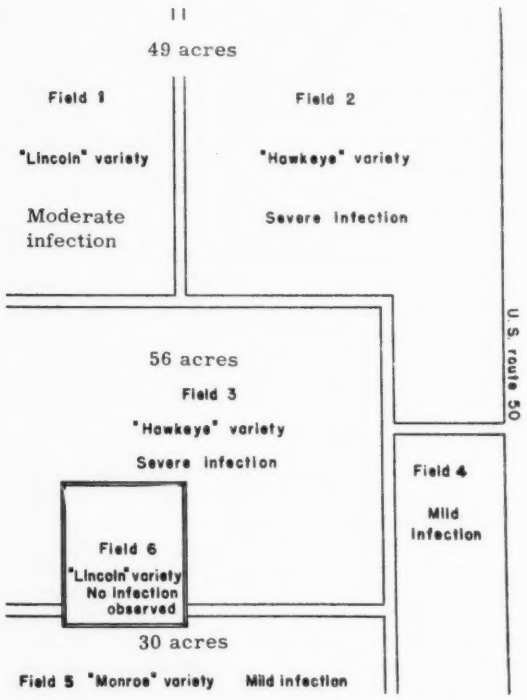

FIGURE 1. Diagram showing the relation of the several soybean fields to each other, field 6 (insert) about 5 miles distant from fields 1 to 5.

On September 20, the senior writer, the grower, and the assistant county agent opened pods in field 2 and found the seed to be small, shrivelled and discolored. Field 1 of the Lincoln variety was examined for the first time. The Phyllosticta disease was readily recognized on pods, leaves, and stems. Stem cankers were even more conspicuous than on the Hawkeye variety (Fig. 2, E and F). The diseased condition of the Lincoln variety in field 1 led to an examination of another field of Lincoln soybeans five miles distant that was sown with the same seed (field 6). This field proved to be healthy. It thus appeared that field 1 of the Lincoln had become infected from the adjoining Hawkeye fields. It is probable that fields 4 and 5, too, became infected from the diseased crop of Hawkeye. Apparently the seed for this crop had been abundantly infected.

The plan had been to sell the seed of the Hawkeye and Lincoln varieties for planting. Field 3 was a total loss. Seed from field 2 was disposed of for low grade feed and oil, at a 50 percent loss. An estimated loss of one-third occurred in the Lincoln variety in field 1.

The pathogen of the disease at Salisbury was determined as Phyllosticta sojaecola Massal. (8). Isolations were made from diseased leaves, stems, and seeds of soybeans collected at Salisbury and from diseased leaves collected in June in a plot at Beltsville, Maryland (Fig. 2, G and H). Inoculations with these cultures on seedlings of Hawkeye in a greenhouse at Beltsville resulted in leaf symptoms identical with those observed in the field. Reisolations of the fungus were made.

Heretofore, in the United States the Phyllosticta disease has been reported as attacking only soybean leaves, although Crall (3) reported a brown streaking of the stem accompanying defoliation. In this case he recognized the pathogen as Phyllosticta glycineum Tehon (11). Atkinson (1) described as a "white stem canker" of undetermined cause which could well have corresponded to the stem canker at Salisbury. An earlier record of the disease on pods, seeds, and stems is that by Böning (2) in Germany. Liu (7) isolated P. sojaecola from soybean seeds in China. According to Ling's observations in China, pod and stem as well as leaf attack are by no means uncommon.[5] In Ontario, Canada, where the disease developed in eipiphytotic proportions in 1943, only leaf attack was noted (5).

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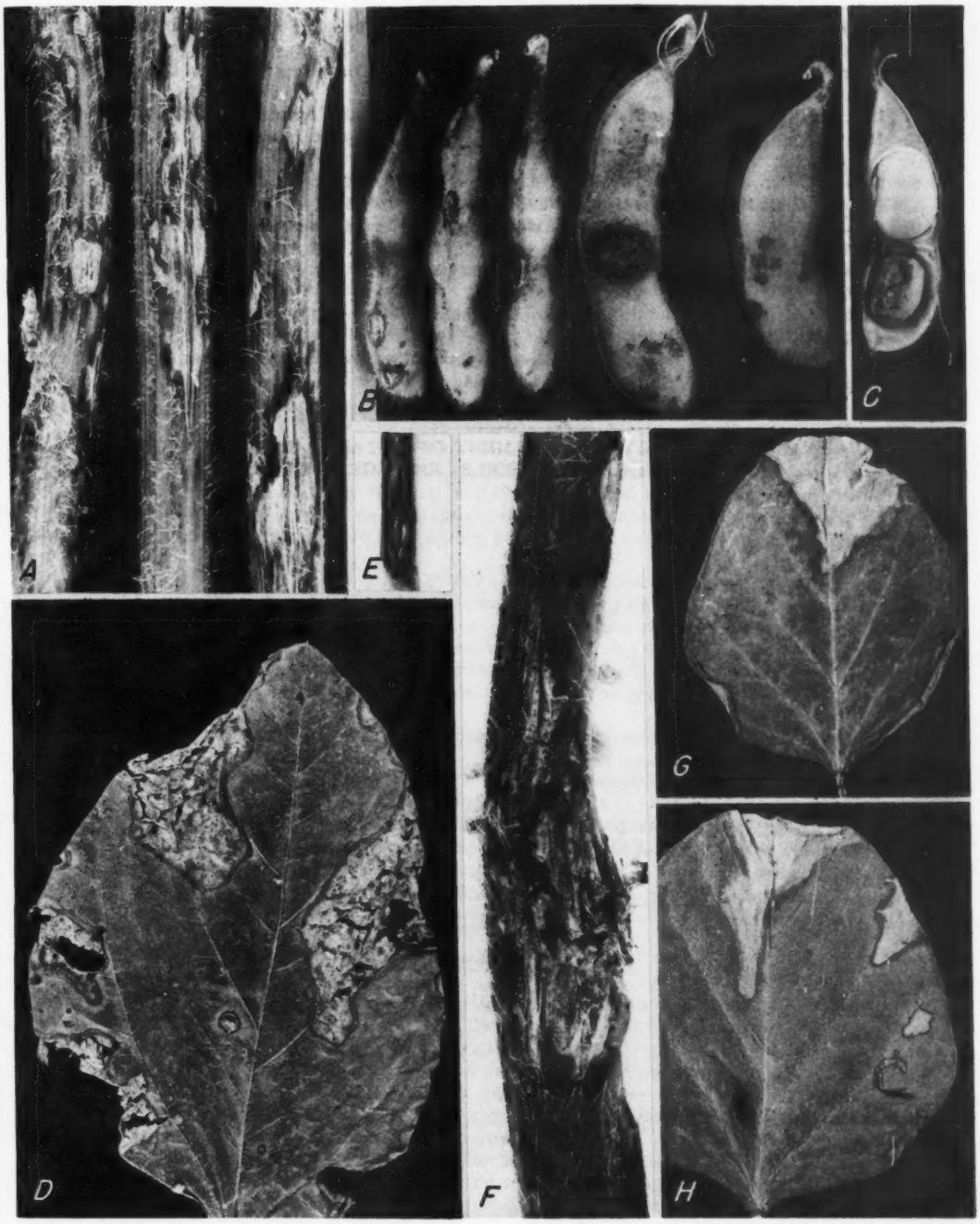

FIGURE 2. A-F Symptoms of the disease as observed on soybeans at Salisbury, August and September 1951. A. Stem cankers on Hawkeye variety, X3. B. Lesions on pods, XI. C. Pod with one valve removed exposing a diseased and a healthy seed, XI. D. Spotting on a mature leaf of Hawkeye, XI. E and F. Stem cankers on the Lincoln variety, E, XI: F, X5. G and H. Phyllosticta spotting on soybean leaves at Beltsville, June 1951, XI. Photographs by Kreitlow (A-D and G and H) and by R. L. Taylor (E and F).

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DIVISION OF PLANT PATHOLOGY, DEPARTMENT OF BOTANY, UNIVERSITY OF MARYLAND, AND U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

A STEM RUST OF PINUS ROXBURGHII IN INDIA POTENTIALLY DANGEROUS TO THE UNITED STATES

Perley Spaulding[1]

In 1890 Barclay (5) stated that he had seen in the vicinity of Simla, India, a single tree of P. longifolia [Pinus roxburghii] with a stem rust, and named it Peridermium complanatum var. corticola, but gave no description of it. This appears to be the first definite mention of this fungus. Through the succeeding years it became known to the foresters of India as the cause of a serious disease in young stands of this tree (3, 6, 7, 8, 12, 13) (Fig. 1).

For about 40 years nothing was known of the alternate host plant of the rust. Then a special search revealed that herbaceous plants of the genus Swertia (Gentianaceae), growing among the ground cover under a heavily diseased stand of pine regeneration, bore abundant telia of Cronartium (Fig. 2).

Inoculations with spores of Peridermium on five species of Swertia growing in that part of India showed that S. alata and S. augustifolia are very susceptible, S. cordata is lightly infected, and S. purpurescens and S. paniculata are immune. Unsuccessful inoculations also were made on species of Quercus, Rhododendron, Myrica, Pieris, Rhus, Randia, Woodfordia, Inula, and Pyrus, all plants growing in or near pine rust outbreak areas (1, 4). The fungus was named Cronartium himalayense Bagchee, with the pine stage Peridermium himalayense Bagchee.

After an interval of some years Bagchee published the results of inoculations made on needles of 180 P. roxburghii trees 2-1/2 to 5 feet tall with telial sporidia of the Cronartium from Swertia. Both juvenile and mature needles became infected, pycnia developing in the bark of twigs 6 to 20 months later, and aecia in the third to fifth spring after inoculation. Most of the infected trees died when aecia were produced (2).

Data on damage to the pines are not available, but there is heavy mortality of young trees locally. Pines more than 25 to 30 years old are said not to be attacked. The youngest die promptly. Older saplings survive for some time as they are not girdled so quickly by the fungus. Large poles may outgrow the disease (2). In one stand of heavily diseased P. roxburghii a group of about 50 trees of P. ponderosa and its variety arizonica (mostly the latter) had been planted some years before, and apparently were entirely healthy, although many of the Indian pines were dying or dead (2).

Discovery of the alternate hosts of the fungus causing the disease made possible the planning and testing of practicable methods of control. These appeared to be limited to (1) substitution of an immune pine for P. roxburghii, and (2) eradication of alternate hosts in the vicinity.

Pinus roxburghii is the main resin-producing tree of India. A substitute that is immune to the stem rust ought also to be suitable for resin extraction. Consideration of the native species seemed to show that there is no completely acceptable substitute among them. This conclusion leads to control of the pine rust by eradication of Swertia. This is difficult because Swertia is a herbaceous plant which grows intermixed with forage grasses and produces an abundance of small seeds. Some attempt was made to eliminate Swertia but no details are available on the results.

Discussion

The detailed description of the disease and illustrations of the fungus convince the writer that this fungus affects the attacked pines exactly as does the white pine blister rust (Cronartium ribicola Ed. Fisch.). Except for the difference in hosts and locale, one could substitute the name C. ribicola for that of the Indian fungus and have the account almost completely applicable. The effect of the fungus appears to be much like that of our native blister rusts on their hard pine hosts. Swelling of the infected bark is followed by cracking where the spore blisters rupture it, and final loosening. Secondary fungi and insects attack it and pitch exudes. Young trees frequently are stunted and resemble dense witches' brooms. The leaves are yellowish and drop earlier than those of healthy trees (1). This rust must be considered a potentially dangerous disease for hard pines in this country until we can learn whether American pines and Swertias are susceptible.

India is known to have a rich rust flora and a number of pine stem rusts. Although much experimental work has been done in determining the alternate hosts of the heteroecious rusts. there is still more to be done, as is also true in our own country where a new stem rust of a hard pine was discovered recently (11).[2] While Pinus roxburghii is the only known pine host for Cronartium

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[2]A fuller account and description of the fungus has been submitted for publication in Phytopathology, by George H. Hepting and George B. Cummins, with the title "A new species of Peridermium on Virginia pine."

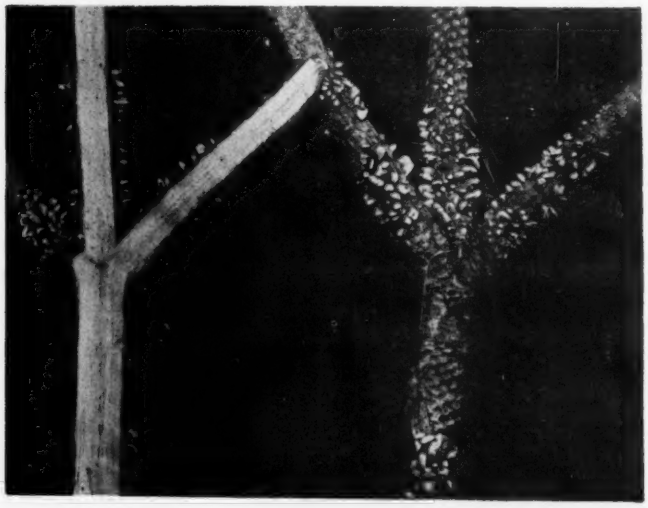

FIGURE 1. Peridermium himalayense Bagchee on Pinus roxyburghii Sargent. Infection on a young sapling, showing aecia not fully opened. X approx. 3/16. Reproduced, with permission, from Bagchee (3).

FIGURE 2. Cronartium himalayense Bagchee on Swertia alata Roxb., showing the telia crowded on the leaves and stem. X approx. 3. Reproduced, with permission, from Bagchee (4).

himalayense and is believed to be the only one, it may be that some other pine is a yet unrecognized host. Strict limitation to one pine host, while not unknown, is rather unusual. If it is found to be true with this rust, it may indicate that American hard pines are not likely to be susceptible, although that must be proved by inoculation experiments. The above-mentioned case of 50 trees of P. ponderosa and its variety arizonica remaining apparently unaffected among heavily diseased P. roxburghii in India is encouraging, but it must be remembered that all these trees have heavy scaly bark early in life which greatly complicates detection of the fungus fruits.

Turning to our American situation as regards Swertias and pines, we find that we posses 20 hard pines (P. roxburghii is a hard pine), including nearly all of our extremely valuable southern species. Their combined natural ranges extend across the country from the Atlantic to the Pacific

Oceans and from our northern to our extreme southern boundaries (9). Pinus roxburghii is a sub-tropical species, but temperature may not be a limiting factor for the rust.

As possible alternate hosts we have 25 species of Swertia widely distributed over the country. The species in the eastern United States ranges from New York and Pennsylvania westward to Wisconsin and southward to Arkansas, Louisiana, and Georgia. The other 24 species have a combined range covering the Rocky Mountain and Pacific Coastal Regions (10, 14). We may have serious trouble if the fungus ever reaches our shores, and if any of our hard pines or Swertias are susceptible.

The only practicable way to learn the reaction of our pine and Swertia species to the rust would be to grow them in an area in India where the rust is present. This seems to be impossible at present, but may sometime in the future become feasible. Such tests might show that our longleaf pine is immune and that it might be used as an acceptable substitute for the susceptible Indian pine.

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DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, NEW HAVEN, CONNECTICUT

OAK WILT SURVEYS IN 1951

Marvin E. Fowler

The discovery of oak wilt caused by Chalara quercina in many new localities and in seven additional States in the summer of 1951 increases the concern already felt about this rapidly killing disease of our oaks. Surveys initiated in 1950[1] were continued on a larger scale in 1951, and it is expected that even more extensive surveys will be conducted in 1952.

Airplane Survey

Scouting for oak wilt from low-flying airplanes has proved to be highly satisfactory because of the pronounced leaf symptoms on affected trees. These leaf symptoms vary with the oak species affected. Bronzing of leaves develops rapidly on trees in the red oak group, usually beginning in the top of the tree and then at the ends of the lateral branches. Trees in this group are usually killed during the same season in which they first show symptoms. The disease develops more slowly on trees in the white oak group, and in newly infected trees the discoloration of leaves is frequently confined to a single branch for an entire season. The following year additional branches may be affected, but the tree may live several years before death occurs. Even the earliest external symptoms on red oaks, that is bronzing of the leaves on the terminal branches in the tree crown, can often be seen more easily from the air than from the ground directly beneath diseased trees. From the ground these bronzed leaves appear as a narrow streak against the sky, whereas from the air they may appear as a brilliant bronze spot in contrast to the green foliage of surrounding healthy trees. Oak-wilted trees can be seen readily from ridge or valley roads wherever a good view can be obtained of the tree crowns; also from a fire tower. Such areas are exceedingly limited, however, and in flat country and areas not visible from roads or fire towers airplanes provide the fastest, best, and cheapest means of surveying the forests for the disease.

In a low-flying airplane, at 500 feet or less above the tree tops, oak wilt can be distinguished with reasonable accuracy. The symptoms of the disease are seldom confused with those produced when oaks are dying from other causes. Even trees that are lightning-struck or trees dying from poisoning can usually be recognized by an experienced aerial observer; such trees may die while in full leaf, with the leaves turning bronze evenly over their entire surfaces. Oaks dying from the wilt have usually undergone partial defoliation and parts of leaf blades stay green for several weeks. The crowns of such trees may appear thinner and of a slightly different color than those dying from lightning or poison.

An observer in an airplane 200 to 500 feet above the tree tops can see a dying oak for one-half mile or more, but his most effective survey for wilt is limited to a strip about one-quarter mile wide. Two observers in a plane can usually survey a strip one-half mile wide through the forest. A third member of a crew can greatly speed the work by keeping the position of the plane located at all times on a map, indicating thereon the location of suspected trees seen by the observers, and describing landmarks that will be useful to a ground crew in visiting the area.

In an area where the wilt has not previously been known, ground crews must collect specimens from suspected trees for fungus isolation to make certain that an accurate diagnosis is obtained.

Distribution of Oak Wilt

Oak wilt was found in many additional localities during 1951. Figure 1 shows those counties in which oak wilt was confirmed by cultures before 1950, during 1950, and in 1951. This wide range of the disease does not in itself give any information regarding the yearly spread of the disease. In many places the wilt was not found until 1950 or 1951 because no search was made for it until that year. Nevertheless, some evidence has been gathered concerning the spread of oak wilt.

Pathologists in various States have made estimates of the age of the oldest oak wilt infection found in some counties. Members of the Division of Forest Pathology have made similar estimates for some areas. It is exceedingly difficult to estimate the age of the oldest infections in those States where the disease has been present for two decades or longer. The estimates that have been made are presented in Figure 2.

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[1] Fowler, Marvin E. Surveys for Oak Wilt. Plant Dis. Reptr. 35:112-118. 1951.

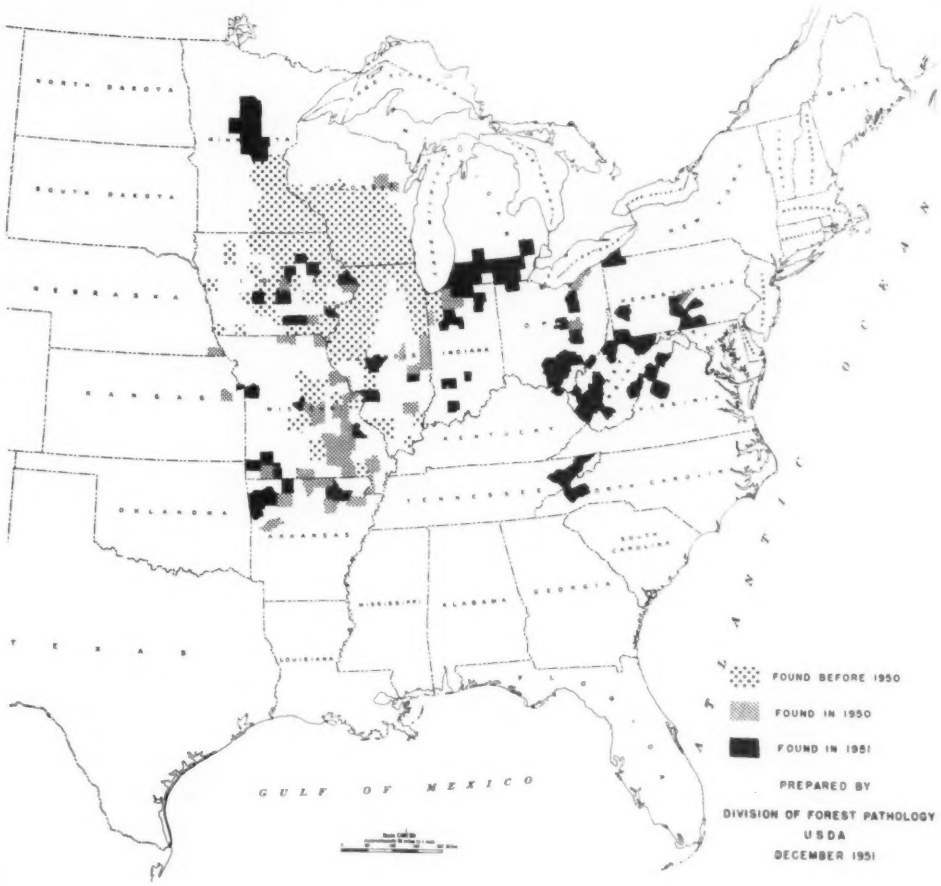


FIGURE 1. Distribution of oak wilt in the United States.

The age of the oldest oak wilt infections in Wisconsin, Iowa, and Minnesota is not known. The disease was first observed in these States, but a cultural means of identifying oak wilt was not developed until about ten years ago. Prior to that time oaks were observed to be dying from an unknown disease. We cannot be certain that the earlier reports were of oak wilt, but a description of symptoms of an oak disease in Wisconsin and Minnesota, written by a forest pathologist in 1912, could easily be applied to a known oak wilt infection today. The size and condition of the larger oak wilt infection centers in these States and in Iowa make it appear that the disease has been there considerably longer than 10 or 20 years. It is therefore reasonable to estimate the age of the oldest infections in Wisconsin, Minnesota, and Iowa as being 20 years or more old. In northwestern Indiana there are a few infections that appear to be at least 20 years old. Possibly there are some of these older areas in northern Illinois and Missouri.

The older infection centers have an entirely different appearance from the more recent ones. The youngest infection centers among red oaks usually consist of a single oak tree expressing foliar symptoms for the first time. A year later this tree is dead and there may be one or several freshly wilting trees in the immediate vicinity. Each year a number of additional trees may wilt and each year the dead trees progressively deteriorate. In five to ten years the resulting pocket of dead trees may be 1/8 to 1 acre in extent and sometimes even larger. The oldest infection centers in Wisconsin are characterised by being much larger and by having an almost barren area in the middle of the infection. Oak wilt kills roots and stump and prevents the usual stump sprouting. In a forest stand that is almost all red oak, reproduction in the oak-wilt killed area must be mostly through natural seeding from outside the area. This process is slow and for years an area

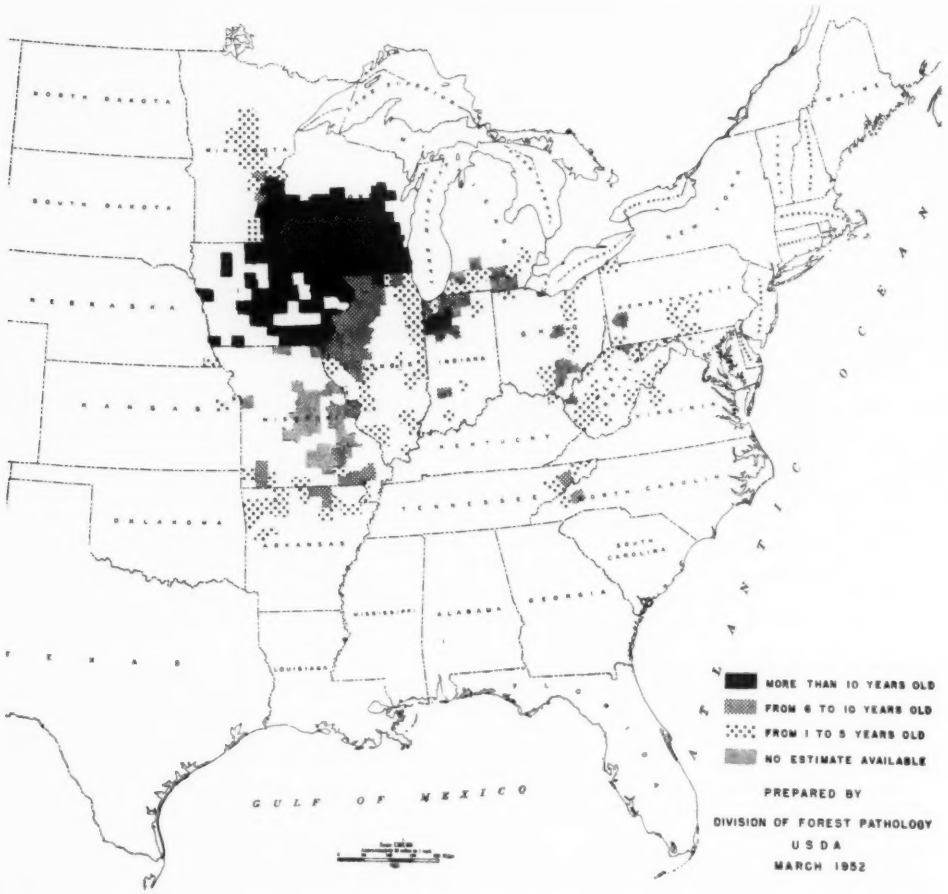


FIGURE 2. Estimated age of oldest oak wilt infections.

almost devoid of trees results. In Wisconsin there are a number of very large infection centers with large barren areas surrounded by standing and windthrown dead oaks in various stages of deterioration; at the outer edge of these infection centers there are oaks dying of the disease during the current season. No areas like this have been found in the Ozarks or in the East. In these regions most of the infections found are young, from one to five years old, and consist of from a single tree to a half dozen trees. In a few counties there are some older and larger infection centers, but as yet none have been found that are estimated to be over ten years old. In a number of counties marked as infected in Figure 1, only one location with oak wilt was discovered.

With very old infections prevalent in Wisconsin, Minnesota, and Iowa, with younger infections in northwestern Indiana, northern Illinois and possibly northern Missouri, and with all the infections found elsewhere being less than ten years old, mostly from only one to five years old, it appears that oak wilt has been spreading southward and eastward.

The total number of infection centers found in the Ozarks, in Michigan, and from Ohio eastward and southward, and the total number of infected trees, are both quite small. Less than 500 oak-wilted trees have been found in Arkansas and east of Indiana. On the basis of present information eradication of all of these infections would appear to be justifiable, to reduce the hazard of spread. However, some spread may occur from them before they can be removed. Subsequent surveys would be necessary to detect new outbreaks that may have originated from them or from some distant point. It is hoped that research now under way may reveal a cheaper and more effective control than the present method of control through eradication. In the meantime, eradication is the best method known to prevent the disease from becoming too prevalent and widespread

to be amenable to control measures.

The Division plans to prepare additional reports on the distribution and prevalence of oak wilt, and will appreciate being informed of outbreaks in new areas.

Surveys Conducted

The Division of Forest Pathology had three oak wilt survey crews during the summer of 1951. One group searched for the disease in southern Missouri, Arkansas, and a small portion of western Kentucky and Tennessee. Another group surveyed in the western half of Mississippi and the eastern half of Louisiana. The third group surveyed in western North Carolina and eastern Tennessee and made a very limited search for the disease in western Virginia, eastern Kentucky, and northern South Carolina. The first two groups used airplanes to search for suspected trees while the last group did most of their surveying from automobiles on ridge and valley roads and from fire towers.

A number of other organizations conducted surveys for oak wilt in 1951. Arkansas cooperated in the survey conducted by the Division of Forest Pathology, by furnishing airplanes for surveys over State Forests. Surveys were continued by State officials in those Lake States where oak wilt has been known for the longest time. Illinois increased survey activities. The Cook County Forest Preserve resurveyed its forest last summer. Texas surveyed approximately 10 percent of its Region 1, consisting of the ten northeastern counties, using an airplane for the preliminary work. Indiana increased its search for the disease and employed one man to devote the summer to this work. Michigan conducted a survey over the three southern tiers of counties in that State, using an airplane for preliminary work. Ohio searched for the disease with a State-owned plane. New York initiated an airplane survey in the southern part of that State. Pennsylvania conducted a limited airplane survey. New Jersey, Massachusetts, and Maryland searched for the disease, and a limited survey was made in western Maryland from airplanes. West Virginia surveyed about 10 percent of the State with an airplane. South Carolina cooperated with the Division of Forest Pathology by furnishing an airplane for several days. Georgia had foresters visit each fire tower in 40 counties to search for the disease. Rhode Island made a complete State-wide airplane survey. The Tennessee Valley Authority scouted several counties in east Tennessee.

A compilation of the results of all these surveys is shown in Figure 1. Oak wilt was found in many new counties and in the following additional States: Michigan, West Virginia, Virginia, Maryland, Kentucky, North Carolina, and Tennessee,

It is hoped that this disease will be brought under control in forests with high values in oaks and that new infections will be eliminated as they appear. Control programs for oak wilt are most likely to succeed if they are initiated while the infection centers are small and not numerous. Eradication of all known oak wilt infections in the Ozarks and Eastern States would not be too difficult now, but if delayed several years the number of infections will probably more than double, and the disease may get completely out-of-hand.

Airplane Versus Ground Surveys

Accurate information that can be obtained quickly is sometimes urgently needed on the distribution and severity of tree diseases. This has been particularly true for oak wilt. The usual method of conducting ground surveys would have been so slow that by the time the survey could be completed its usefulness as a guide for a control program would very likely have expired.

By using airplanes for a large portion of the work, information on the distribution and severity of oak wilt has been made available in a short time. The use of airplanes is also much cheaper than other survey methods. With a three-man crew in an airplane, two observing and the third handling maps, a one-half mile strip can be surveyed through the forest. After allowing sufficient loss of time to indicate accurately the location of suspected trees on a map, these three men can survey about 32 square miles (20,480 acres) of forest land in an hour. The same number of men conducting a thorough cruise on the ground could follow a compass line about 4 miles in a day and survey an area about 2 chains wide, which would be 64 acres. It would, therefore, require about 320 working days for these three men to cruise a forested area on the ground that they could survey from an airplane in one hour. With surveys for a disease like the oak wilt, which must be restricted to the summer months when foliar symptoms are pronounced, this would require several years.

DIVISION OF FOREST PATHOLOGY, U. S. PLANT INDUSTRY STATION, BELTSVILLE, MARYLAND

BRIEF NOTES ON PLANT DISEASES

TWO-YEAR OLD PLANTING OF MONTEREY PINE KILLED BY LOW WINTER TEMPERATURE

By L. W. R. Jackson

In March 1949, two thousand 1-year-old Monterey pine (Pinus radiata D. Don.) seedlings were planted on the Georgia Forestry School forest at Whitehall, Georgia. The planting stock had been grown in the State nursery at Flowery Branch, Georgia. The planting survival was 85 percent. Establishment and shoot growth for the 1949 and 1950 growing seasons were satisfactory. The entire planting was killed during the 1950-1951 winter season. The loss of the planting is attributed to the unusually low temperatures of 8°F. on November 25, 1950 and 9°F. on February 3, 1951.

SCHOOL OF FORESTRY, UNIVERSITY OF GEORGIA

A NEW RACE OF HEAD SMUT ON BROMAR VARIETY OF MOUNTAIN BROME

By Jack P. Meiners

In the summer of 1950, heavy infestations of head smut (Ustilago bullata) were reported in fields of the Bromar variety of mountain bromegrass (Bromus marginatus) in seed producing areas of Eastern Washington. As this variety had previously been resistant to head smut, it was suspected that a new race of smut might be present which is capable of attacking Bromar. Subsequent inoculation experiments have shown this to be true.

Smut collections were secured from fields of Bromar and from harvested lots of Bromar seed. Seed of Bromar was inoculated with these smut collections and grown in the greenhouse. All inoculations resulted in very high (76 to 100 percent) infection. Bromar inoculated with these same collections and grown in the field in 1951 was 100 percent smutted.

Bromar mountain brome was released primarily because of its resistance to head smut, to which other mountain brome varieties and selections are highly susceptible. Artificial inoculations of Bromar with races of head smut had resulted in only very small percentages of infection and in commercial plantings it has been smut-free until very recently. Now, however, the recent heavy incidence of head smut in field plantings and the results of the inoculations described above indicate that Bromar is highly susceptible to a new and a very virulent strain of head smut. Although Bromar is extremely susceptible to this new race, it is still superior to the common strains of mountain bromegrass in that it is resistant to the other four races of head smut which attack Bromus marginatus and related grasses. However, since Bromar is now subject to attack by the new race it is imperative that seed of this variety be treated if the incidence of head smut is to be held to a minimum.

The exact relationship of this new strain of head smut to the established races of head smut is under investigation. A search has also been initiated to find other sources of resistance to head smut in mountain brome.

DEPARTMENT OF PLANT PATHOLOGY, AGRICULTURAL EXPERIMENT STATION AND DIVISION OF FORAGE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, U. S. D. A., PULLMAN, WASHINGTON, COOPERATING

DISEASES OF SORGHUM AND CORN IN NIGERIA, AFRICA, 1951

By O. J. Webster

The writer spent about nine months in West Africa in 1951, organizating research projects in corn and sorghum improvement. While traveling over this region, careful observations were made on the prevalence of corn and sorghum diseases, and typical specimens were collected. These were later identified by R. W. Leukel, Pathologist in Cereal Diseases at the Plant Industry Station at Beltsville, Maryland, and deposited in the Mycological Collection at that station.

Sorghums have been grown in West Africa for centuries, and no doubt natural selection has been very effective in the development of varieties resistant to various sorghum diseases. Diseases are not so serious a problem in sorghum production in Nigeria as might be expected considering the warm humid weather conditions that prevail during the growing season. The extremely dry season of five or six months between crops, when the temperatures usually reach the 100-

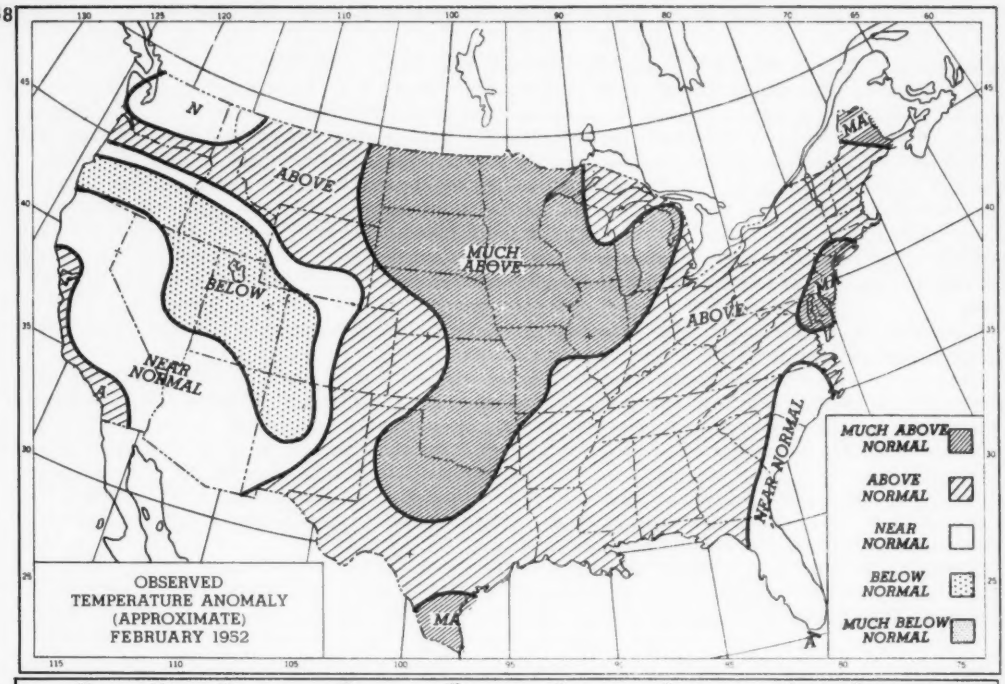
degree mark or higher each day, may limit the survival of the inoculum.

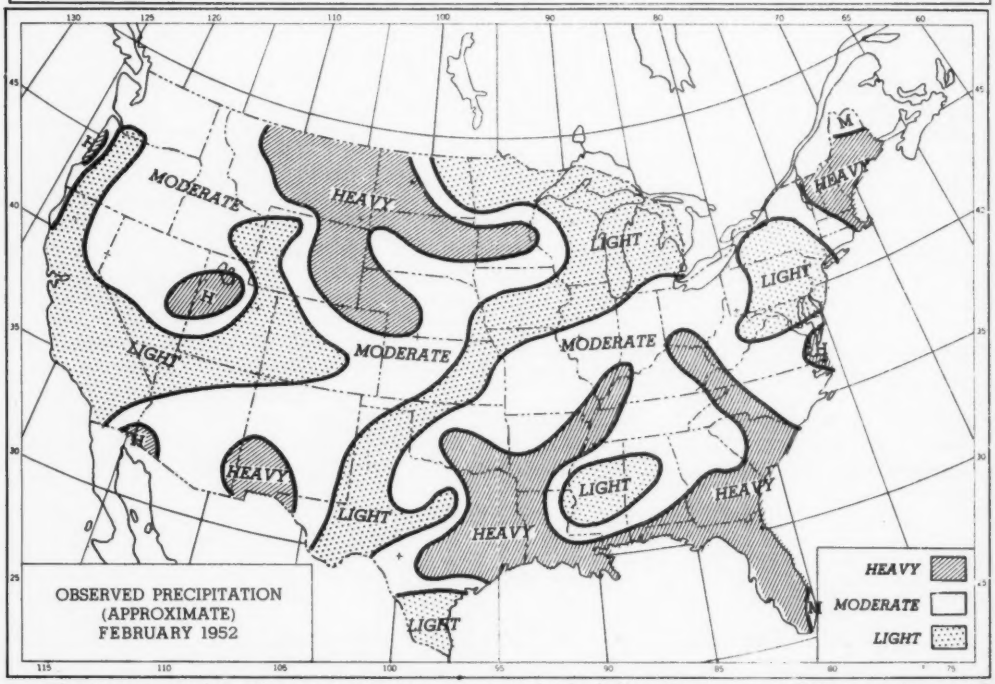
A collection of sorghum varieties from Turkey and India were nearly defoliated by sooty stripe (Ramulispora sorghi). The local varieties and a collection common to the United States showed a high degree of resistance. Anthracnose (Colletotrichum graminicola) and zonate leaf-spot (Gloeocercospora sorghi) were found on the leaves of several varieties late in the season. Bacterial stripe (Pseudomonas andropogoni) was the most prevalent leaf disease of the local varieties but the injury was slight. Covered kernel smut (Sphacelotheca sorghi) was commonly found in farmers' fields, and infection ranged from a trace to 20 percent of the heads. Some smut was found on plants grown from seed treated with copper carbonate or Spergon. It is possible that the smut spores survive in the soil. Seed treatment is not generally practiced by the farmers.

Sorghum rust was found in trace amounts in Nigeria, and charcoal rot was very prevalent in large fields of feterita in the Anglo-Egyptian Sudan.

Corn is an important food crop in southern Nigeria and Gold Coast. In 1950 and 1951, corn rust caused serious losses to the crop. The buildup of rust in these 2 years was first thought to be due to a new race of Puccinia sorghi but, late in the summer, specimens collected along the west coast of Africa were identified as P. polysora. This species has never before been reported in Africa. Funds have been appropriated by the Colonial Office in London for the improvement of corn in Nigeria and Gold Coast, and much of the effort will be directed towards breeding varieties resistant to rust. Some material at Ibadan, Nigeria, showed sufficient promise to merit its consideration for use in breeding for rust resistance.

DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, AND NEBRASKA AGRICULTURAL EXPERIMENT STATION, LINCOLN, NEBRASKA





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.